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VETERINARY MEDICINE

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No. 6

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Most hoofed animals have horizontal eye pupils.

* * * *

A combination of sulfanilamide and dinitrophenol is said to prolong the life of guinea pigs infected with tuberculosis.

* * * *

The rabies situation in Michigan is running wild.—*State Dept. of Health.*

* * * *

Dried prunes contain vitamin A and all known members of the vitamin B complex. —*S. N. L.*, 37:16.

* * * *

Ochsner and Smith report that vitamin B_1 (thiamin chloride) is effective in relieving the pain of varicose ulcers.

* * * *

A fifteenth century writer described a greyhound as a dog with "the head of a snake, the neck of a drake, the foot of a cat, the tail of a rat, the side of a bream, and the back like a beam."—*S. N. L.*

* * * *

The fifth revision of Merck & Company's "Nicotinic Acid" has been issued. This is a compilation of available data on nicotinic acid—its structure, history, function, disease conditions caused by its lack and their treatment, etc.—with a bibliography of the literature to date, including 117 titles.

Surgery may not require brains or bring a fortune, but it is the best job in the world. —*Doctors in Shirt Sleeves.*

* * * *

One grain of gramacidin, a newly-discovered chemical produced by soil bacteria, will protect a mouse against 10,000 fatal doses of pneumococci or streptococci.

* * * *

Lemonade was sold as a scurvy remedy by medieval pharmacists in Florence, although not until recently has science understood the vitamin C value of citrus fruits.

* * * *

The new synthetic vitamin K is soluble in water. The natural vitamin K is not. This increases its efficiency as a hemostatic agent, since it can be administered intravenously and thus is available in emergencies.

* * * *

There were no human deaths due to rabies in Detroit in 1939, and only 80 dogs were reported as rabid. Detroit licensed 58,739 dogs; of this number 15,075 (over one-fourth) were vaccinated against rabies. The Pasteur treatment was administered to 1,330 persons bitten by dogs "believed to be rabid."

The Tovrea Packing Co., Phoenix, Ariz., has purchased the 275,000-acre Double O ranch in Yavapai County, Ariz. It is expected to run 7,000 yearlings and two-year olds on the ranch, moving them to the company's valley ranches for conditioning before pen-feeding.

Libby, McNeill and Libby, meat packers, made a net profit of $3,782,236 in the fiscal year ending March 2, 1940. In the preceding year the firm operated at a loss of $3,354,279. The company's sales for the year were $73,080,185.

The quantity of lard in storage reached an all-time high of 269,284,000 pounds April 1, an increase of 12,644,000 pounds during March. The normal stock of lard runs around 140,000,000 pounds. Pork holdings stood at 653,552,000 pounds on the same date —73 million pounds above the five-year average. However the consumption of lard is running 15% above that of a year ago.

The present Canadian import quota for pork from the United States provides for a maximum of 1,647,000 pounds monthly. The Anglo-Canadian bacon agreement provides for weekly shipments of 4,480,000 to 5,600,000 pounds of bacon and hams. The number of swine in Canada Dec. 1, 1939, was 34% larger than it was a year earlier. A further increase is expected this year.

Statistics on Live Stock Slaughter

The following comparative figures, reported by the U. S. Department of Agriculture, show the number of animals slaughtered during January and February in all plants in the United States under federal inspection, for the last seven years:

	Cattle	Hogs	Sheep
1940	1,542,466	9,633,005	2,910,734
1939	1,414,027	6,933,580	2,816,675
1938	1,545,949	7,034,034	2,975,000
1937	1,575,686	6,361,910	3,015,309
1936	1,647,869	5,746,895	2,853,657
1935	1,636,005	5,455,859	2,481,509
1934	1,563,994	8,824,359	2,565,763

In February packers slaughtered more hogs—4,277,212—than during any February in the last eight years.

Florida's Tropical Cattle Tick Long Known

Confidence in the eventual eradication of cattle fever ticks, which are responsible for tick fever, was voiced by Dr. John R. Mohler, Chief of the Bureau of Animal Industry, U. S. Department of Agriculture, in discussing the problem of tick-infested deer in central Florida. Doctor Mohler, who recently returned from the area, said that the tick-eradication campaign is making normal gains and is preventing reinfestation in other areas. "Throughout the South," he added, "public sentiment, in general, is still strongly in favor of the elimination of the last tick, including the Australian or tropical variety which infests deer as well as cattle."

In the early days of eradication work, Doctor Mohler also pointed out, the veterinarians had many obstacles to overcome, some of which were much more serious than the tick-infested deer in a small part of Florida. Commenting on the opposition of persons who object to the killing of tick-infested deer in parts of two counties to hasten the progress of tick eradication, Doctor Mohler disavowed claims that this phase of the work was unforeseen and also statements to the effect that the Australian or tropical variety is a recent discovery.

This variety of tick was found in Florida over 28 years ago. In 1933, Doctor Mohler reported that reinfestations in tick-free areas were apparently due to the presence of numerous deer and the Australian variety of tick.

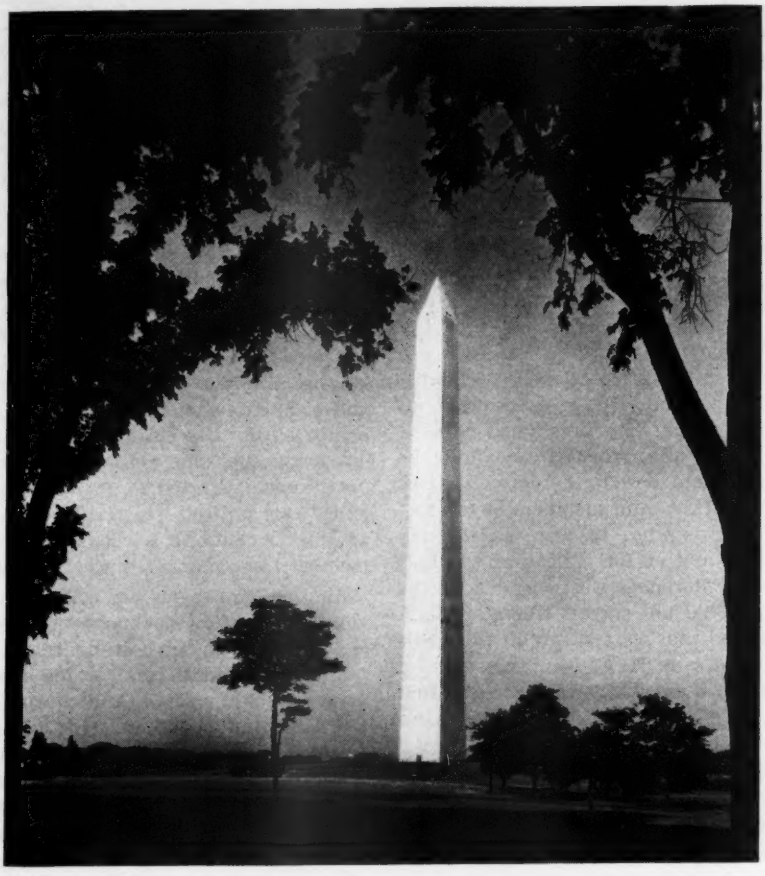
The tropical cattle fever tick, *Boophilus annulatus* var. australis, is widely distributed throughout the tropical and sub-tropical countries of the Western Hemisphere. The habits and life history of this variety of cattle fever tick are essentially the same as the North American cattle fever tick, which it very closely resembles. The outstanding structural difference is a prominent protuberance on the posterior margin of the male. The females of the two varieties of tick resemble each other very closely. Both varieties are capable of transmitting tick fever in cattle.

A. V. M. A. Dog Food Program Needs Revision

A reinvestigation of its dog food program should receive careful consideration at the Washington meeting of the A. V. M. A. The present program has led to dissension among members, to charges of racketeering, to resentment among the manufacturers of the better dog foods and it must be said is not working—yet. Surely a program the objects of which are so laudable can be so formulated as to receive general approval and support.

Over the long period of its history, policies established by the A. V. M. A. have been sound, with but few exceptions. It has rarely needed to reverse itself because of hasty action. The action taken at the Memphis meeting on this matter was an exception to the rule in that it was not only hasty but taken with little information available to three-fourths of the members of the House of Representatives present, who first learned of it when the project was presented to them for approval.

Furthermore, some of the information



WASHINGTON MONUMENT

Probably none who attends the 77th Annual Meeting of the A. V. M. A. in Washington, D. C., next August will fail to visit this shrine, which is known to more persons and more often photographed than any other in this country.

that was given to the assemblage in response to questions of delegates was materially incorrect. For instance, it was stated and more frequently implied, that this project of the American Animal Hospital Association was succeeding and was "preeminently doing a good job." The fact is that in the previous four years the project had enlisted but three out of some 200 or more manufacturers of commercial dog food and that none of the three nor all of them together constitute a material factor in the dog food industry either in the amount of food produced or in the extent of its distribution. Only one manufacturer, and again a comparatively small and unknown manufacturer, has procured a seal of approval since the Memphis meeting. The project was and is to this date a complete flop so far as improving the quality of dog food is concerned. It was stated that the charge to the manufacturer was $500 per year, whereas the price asked is $500 with the application and $450 per year additional (*i.e.* $950 per year) if permission is granted to use the seal of approval. It is significant that official and designate representatives of the A. A. H. A. and members of the Executive Board, who, it was stated, had studied the matter for a year and a half and held conferences with officials of the A. A. H. A., did not correct this statement. It was stated that cooperating in this project with the A. A. H. A. would not entail a charge upon the A.V.M.A. treasury and that the A. A. H. A. guaranteed the national association against harm from damage suits if they should occur, and was willing to furnish bond to this end. No bond has been supplied and the project has already cost the A. V. M. A. treasury a substantial sum for which it has not been reimbursed. Delegates were told that the cost of testing was approximately $500, but no details as to expenditures were given and the additional $450 was not mentioned. This secrecy is responsible for suspicion that has developed since the Memphis meeting. A telephone call to the central office of the A. V. M. A. as this is written elicits the response that that office has not seen the contract submitted to prospective seal holders and has no information as to the price charged for a seal, other than the cost given at Memphis.

Additional misleading statements can be mentioned; however, regardless of all the foregoing, which are more or less matters of opinion, the fact remains that the present setup for certifying dog food is fundamentally wrong. At least one committee of the A. V. M. A.—the Committee on Public Relations—puts a great deal of effort into its endeavor to convince public health authorities, municipal officials and the public generally that the veterinary profession is the logical group to be charged with the inspection of food for human consumption. How much is this negatived by the association going outside the profession for its experts when it undertakes the inspection of dog food? There is no representative of the veterinary profession on the scientific council which passes upon the merits of dog foods for which a seal of approval is sought. And don't think this hasn't been noted and commented upon by public health authorities clear to the top. This is not to question the ability of the eminent nutritionists selected to evaluate dog foods under this project. They are well qualified, as able as could be selected from our own membership—and not one whit abler.

Another weakness, but unlike the foregoing a harmless weakness of the project, is the failure to set a standard, at least a minimum standard that a dog food must attain to be approved, and to approve not the label but the food. This is an utter futility. Note that the seal of approval reads: "American Animal Hospital Association [American] Veterinary Medical [Association] Tested and Approved as Represented on Container. AAHA in cooperation with AVMA." The federal trade commission is charged with preventing unfair statements on the label. The Food and Drug Administration is charged with preventing misleading and false statements on the label. No one has accused either of neglect or incompetence, nor have the federal courts failed in their duty in the matter. For a non-official organization to undertake to duplicate the work of these official bodies is at best, to put it very politely, a work of supererogation.

The result has been just what is to be expected from a meaningless gesture. The great commercial organizations engaged in the production of dog food have not supported the plan. Yet no one of them can be charged with unfriendliness or indifference to the veterinary profession or to the A. V. M. A. All have given ample proof of an exactly opposite attitude. Wilson and Rath, for example, have spent for research into canine nutrition and the improvement of their formulæ far larger sums than the fees charged for approval, having had the work done in leading universities. Swift, Morrell, Chappel and Armour have spent thousands of dollars in their own or independent research laboratories for the same purpose; Gaines, Security, Nutrena, Vitality, Tioga and other dry dog food manufacturers each have expended large sums with independent laboratories for research and improvement of their products. This work has been performed by nutritionists of the highest scientific standing; largely by veterinarians. And, most significant of all, these companies have published their findings, have made available to the veterinary profession the exact nutritional value of their products as determined by scientific biological tests. They have frankly sought the patronage and support of the veterinary profession on the ground of merit alone.

Can as much be said of seal holders? Have any of them taken the veterinary profession into their confidence and in a general mailing detailed the results of scientific research into the nutritional value of their products? Veterinarians as scientific men, informed in nutritional matters, are capable of evaluating scientific research for themselves, and are entitled to complete information about a product when they are asked to endorse it. Under the present A. A. H. A. project all details revealed by the tests are closely guarded secrets, disclosed only to the manufacturer. Members of the association are told only that the food is approved as represented on the container, regardless of how little may be revealed by the statements thereon.

It is not strange that an overwhelming majority of veterinarians have accepted frank and detailed reports of nutritionists and that the sales of the dog food manufacturers who have demonstrated to the veterinary profession and to individual veterinarians the merits of their products, occupy the top brackets when the amount of sales is computed.

As stated in the beginning, this whole matter needs reëxamining at the Washington meeting. There must be a better way. A charge for approval, or an apparent charge for it, is repugnant to the very thought of unprejudiced judgment, however consistent with probity it may be.

* * * *

International Veterinary Congress Accounts

According to Section 49 of the by-laws of the International Veterinary Congresses, accounts of the previous congress shall be settled within two years and shall be published in the veterinary journals of those countries from which the largest numbers of members have been drawn. The following abstract of the accounts of the XIII Congress, held at Zurich-Interlaken in 1938, was presented by the financial committee as of March 13, 1940:

Revenues		
Donations from authorities, firms, veterinary societies and private persons	frs.	71,180.00
Subscriptions of members	"	56,960.90
Total	frs.	128,140.90
Expenses		
General secretary-office	frs.	32,694.94
Handbook, report of the congress, and printed matters	"	52,338.40
Expenses of the congress, including exhibition	"	33,132.56
Contribution to the permanent committee of the congresses	"	1,500.00
Total	frs.	119,665.90
Surplus	frs.	8,475.00

The Swiss Veterinary Society, Emmenbrücke, examined and accepted these accounts at its general meeting January 21, 1940. The surplus was transferred to the Swiss Veterinary Society March 13, 1940.

Going to Practice?

By E. T. BAKER, Moscow, Idaho

AFTER the young veterinarian has passed his final examinations, the following questions confront him:

1. What line of work shall I go into?
2. If in practice, where shall I locate?
3. What equipment shall I need?

The veterinary profession today offers a wide field of activities, and, as youth is the proper time to acquire training and experience, he need not jump into practice at once.

The greatest peril confronting the young veterinarian of today is the fast and expensive mode of living. This is not so much the individual's fault, as the present day system of existence, which requires all one makes, and then some. It is difficult for a young man and his wife to live on a more modest scale than their friends or neighbors. For that reason, when one has a check coming in each month he is loath to give it up for the more precarious receipts of a newly born practice.

■ In choosing a location, one should look for a place which has enough live stock to support a practitioner, with the owners accustomed to calling expert assistance, and able to pay their bills. As the old man said: "They ain't no use fishin' where they ain't no fish."

In one community, about 50 miles from here, there is a well-to-do neighborhood and small town. It is surrounded by a fertile country, with much live stock and well conducted ranches. Driving through this section, one would naturally conclude it to be a fine place for a general country practice.

However, the parents of most of these people homesteaded in this region before there were any veterinarians available. They had to treat their live stock when ailing, and they developed old-fashioned remedies which were handed on from one generation and family to another. Most of these present day farmers have been born, raised, married and live on the "home ranch," and they never think of calling a veterinarian for anything. If the patient happens to live, it is due to their wonderful skill; if it dies, it is an act of God. Half a dozen competent men have located there one after another in the past 25 years, but they have moved away, for the practice there is just nicely adequate for the support of a cigar-store Indian in the manner to which he is accustomed.

In another community, about 20 miles away, a practitioner has lived and prospered. His people call him as a matter of course. They depend on him like our parents trusted the old time family physician. That is what we mean by "good" and "poor" locations.

■ A practice may be acquired in one of the following ways:

1. By locating in a town where there is no practitioner. This may be termed "developing a new field." That is getting it the hard way.

2. By locating where there is already another veterinarian. If the latter is competent and well liked, and there is not enough work for two men, this may be considered as an "uphill proposition." The new man will be given the privilege of "doctoring" for all the deadbeats and slow-pay owners, and will be kept quite busy at first. Later on, when he tries to collect his bills, he will be still busier, then wake up.

3. By purchasing an established practice from a man who wishes to retire, or from the estate of a deceased veterinarian. If the practitioner who wishes to retire has made a success of the business, it will usually be a good investment. If, on the contrary, the present owner is disgusted with collections and the type of work, why do you think you will do any better?

4. By entering into a partnership with another man or old friend. This is one of the best ways to "bust up" a beautiful friendship, and is one of the last types of work we would engage in.

5. By entering the business as an assistant to an older man who wishes to retire in the near future. This is often one of the best opportunities for a young man, for it offers a splendid post-graduate course in conducting a practice. He will learn many things not taught in colleges or mentioned in text books. This training will prove invaluable to him throughout his entire career.

6. By taking over the practice of an old and well fixed veterinarian who wishes to quit and has nothing to sell but some equipment. This is almost like inheriting the business.

7. By making a contract with some farm bureau to care for a number of animals at a specified contract scale of prices. This is socialized veterinary medicine, and in the future, when all agriculture is regimented, and the farmers are paid so much for not planting crops, perhaps the veterinarian will be paid for not making calls. This would be better than not being paid for making calls, when you run into clients like the Scotchman whose doctor told him the bill was three and a half dollars—a dollar and a half for the medicine, and two dollars for the call, and who replied: "Well, Doc, here's a dollar and a half; I'll return the call some time."

■ As to equipment, one can begin on a modest scale and gradually add the instruments and other requirements as needed. A small, compact supply of well made instruments, supplies, medicines and biologics will be found more useful than a cumbersome outfit of everything a high pressure salesman has slipped on the unsuspecting novice. We might add that a pair of good arms and hands; professional judgment and a trace of brains, combined with a pleasing personality, will be found useful in a general practice.

Another pitfall to avoid is the "joining" of every club, lodge and other organization in the town. We could belong to practically every outfit in our town but the Mother's Club, and we could be so busy attending to other people's business we would have no time for our own. Of course, if one is a "natural-born jiner," and likes to be a lead-

General practice brings many "puzzlers." This animal was treated for septicemia until a bacteriological examination revealed the presence of malignant edema organisms.

ing citizen, that is his God-given right as a free and independent American.

■ Expenses will be found fairly high in practice, and one should figure on the net income instead of the gross. Years ago we made the astounding discovery that saving five dollars was equal to making that amount. We also learned that what we made was immaterial as compared with what we collected. We are publishing these amazing discoveries for the first time, for the benefit of humanity in general and the veterinary profession in particular. Perhaps we will be given a leather medal for these scientific researches into the realm of everyday economics—who knows?

Fees vary greatly in different sections, as well as do the methods of collections. No two practices are alike, and no set rules can be laid down for conducting all of them. The practitioner becomes a rugged individualist, and, being one, ourself, we respect the other fellow's ideas along this line.

■ Practice teaches one to be human and kind and sympathetic. The country practitioner sees so much of life and the human element that he becomes tolerant and mellow. He sees poverty and riches, prosperity and depression, and cases die that should get well, and *vice versa*.

There is nothing prosaic or routine about practice. While we have been writing this article, or trying to, we have treated a little fox terrier matron with four puppies for eclampsia; we made a hurried trip to see a cow dying with plant poisoning, and a pure-bred Shropsnire buck suffering from retention of the urine. Next, we treated a dachshund for strychnine poisoning; replaced a uterine eversion in a cow, and injected a "sweenied" shoulder. Then we inspected the meats at seven markets, and examined 42 carcasses of dressed hogs at the local slaughterhouse. Several farmers called for medicine, one for a sick horse and another for some worm medicine for pigs. We answered nine inquiries for a farm journal, listened to a ball game over the radio, and finally got mixed up in a bowling match at the Elks club.

No, one will never get into a rut conducting a practice. It has its drawbacks, but it appeals to us, and that is what counts, from a personal viewpoint.

Ruptured Stomach

Following flatulent colic during harvest, this animal died about fifteen minutes after this picture was taken. A gastric rupture was found during the autopsy a few minutes later. Although we were called as soon as the animal was discovered sick, and we arrived within half an hour, the damage had been done.

This colt was premature, and lived only a few minutes. The dam, a part Arabian mare, was apparently so disgusted with her first offspring that in an attempt to clean house completely she developed a complete prolapse of the uterus. When I answered the owner's frantic call, I found the uterus lacerated in a half-dozen places and covered with manure. After careful cleansing and an epidural block of 2% procaine, the uterus was replaced. A paste consisting of two parts of iodoform, one part of bismuth subnitrate, and enough liquid petrolatum to make a thin paste was inserted and smeared as nearly as possible all over the uterine walls. As a precaution, three loops were placed in the skin on each side of the vulva and the opening laced shut. Antitoxin was administered, and the owner warned that the prognosis was very doubtful. The mare, however, made an uneventful recovery —one of the surprises that make general veterinary practice so interesting.—*H. D. Rodabaugh, Kirksville, Mo.*

Posterior Paralysis in Mares

Within the past year I have seen three cases alike as to history, species, sex, symptoms and outcome. I was unable to make an intelligent diagnosis or give effective treatment in any of them. All were mares. They had been shipped into this section a few days before going down. In each case the attendant reported that they were apparently well at night, but were down paralyzed when seen early the next morning. One was alone in the stable. All had posterior paralysis. At first they attempted to stand. If assisted to their feet they went down again. Within three or four hours all went into a coma, dying within 12 to 24 hours. Respirations, pulse, temperature and visible membranes were normal. Apparently they suffered no pain. Strychnine and calcium gluconate were administered without benefit, except in one case in which the latter gave a slight trembling and a partial return of consciousness. I don't know that this resulted from the medication.—*C. S.*

Sulfapyridine in Calf Septicemia

Since my results with sulfanilamide in the treatment of calf septicemia have been nearly 100% failures, I decided to try the newer drug, sulfapyridine. Although the results were not phenomenal, they bear reporting, if for no other reason than as an indication of the proper dosage of the drug. Gibbons[1] reported "almost miraculous" results with this drug, but I found his dosage entirely too high.

Case No. 1

Subject.—Purebred Jersey calf, three weeks old.

Symptoms.—Respiration labored; feces light yellow and foamy; temperature 104.5°F. No coccidia on microscopic examination.

Treatment.—The diet was reduced to maintenance amounts of dam's milk, and 100cc homologous anti-hemorrhagic septicemia serum was injected into the axillary space. The usual liquid intestinal antiseptics (carminatone, therapogen, āā) were administered. In 24 hours the feces appeared normal, but the temperature was 105.4°. Another 100cc of serum was given, and the intestinal antiseptics were continued. The next day the calf seemed normal except for the appetite, which was very poor, and a temperature of 106.4°. On the following day, there was practically no appetite and the temperature was 106.9°. By this time the calf was very weak, and death appeared imminent. Five grams of sulfapyridine was given as a drench in water, and 12 hours later the temperature was down to 106°. Another 5gm was given, and in another 12 hours the temperature was 103.6°, with the calf much stronger, and starting to eat. Two more 5gm doses were given, making a total of 20gm administered, after which the calf was entirely normal, and has so remained.

The following three cases were all on one farm, where the sanitation was not in keeping with the quality of the calves.

Case No. 2

Subject.—Purebred Guernsey calf, two weeks old.

Symptoms.—Weakness; temperature 104.2°F.; feces white and foamy. No coccidia.

Treatment.—The usual intestinal antiseptics were administered. The diet was reduced one-third in volume, and composed of two parts of dam's milk and one part of lime water, freshly prepared. The next day the calf was still weaker, with a temperature of 103.8°. There was complete anorexia, and the feces were still white and foamy. Sulfapyridine was administered at the rate of 5gm every 12 hours, for a course of 20gm. After the course was finished, the calf seemed normal in every way except for great weakness. In spite of a normal appetite, the patient died 48 hours after the last dose, and only a few hours after a hearty meal.

Case No. 3

Subject.—Purebred Guernsey calf, three weeks old.

Symptoms.—Same as Case No. 2.

Treatment.—Intestinal antiseptics were given, and the diet reduced as in Case No. 2. An injection of 100cc of homologous anti-hemorrhagic septicemia serum was given, and sulfapyridine was administered in the same dosage as in Case No. 2. After the last dose, the calf seemed normal, with good appetite and normal feces, but again it was very weak. Three days later the patient had returned to the same condition as when first examined. The entire treatment was repeated. After completion of the second course of sulfapyridine, the calf was normal except for great weakness. In spite of a ravenous appetite, it died 48 hours later.

Case No. 4

Subject.—Purebred Guernsey calf, two weeks old.[2]

Symptoms.—The same as Nos. 2 and 3.

Treatment.—Intestinal antiseptics were given and the diet reduced as in the preceding cases. Because of the poor results from sulfapyridine in the two previous cases, I tried sulfanilamide in this case, giving three

[1] *Cor. Vet.* 30:1, p. 47.

[2] This and another calf born the same night were given the same prophylactic care. When two weeks old, this one became ill; the other, nearly two months old, is still entirely well. The owner had been instructed in none too gentle terms concerning sanitation, and had been provided with anti-calf scour bacterin, with orders to give 5cc at 48-hour intervals for four doses. The navel and feet were disinfected at birth with equal parts of formalin and water.

60-grain tablets the first day and two the second day, since I feel that 120 grains daily is the maximum dose after the first day. By the evening of the second day the owner reported that the calf was so weak that it appeared it would not live until morning. I drew 500cc of the dam's blood, quickly defibrinated it, and injected it intravenously. The sulfanilamide was stopped as of no value in this case. In 12 hours the calf was much stronger, with a fair appetite, but 12 hours later it was back in its original condition, with a temperature of 104.2°F. I decided to give sulfapyridine another trial, on the following schedule: 1st and 2nd doses, 5gm each; 3rd dose, 4gm; 4th dose, 3gm; 5th dose, 2gm; 6th and subsequent doses, 1gm. After 10gm had been given the calf had a good appetite and the temperature was normal, but it was quite weak and had developed symptoms similar to those of cerebral meningitis. It frantically chewed areas on its body and twisted and turned in dizzy circles for about 15 minutes, then fell exhausted, holding some portion of its skin in its mouth, in a few minutes repeating the entire performance. To relieve the toxic symptoms, one-half teaspoonful of sodium bicarbonate was given every two hours. This worked well, only three doses being needed. The sulfapyridine was continued according to the schedule. The symptoms of toxicity did not recur, and after the one-gram dose had been given four times, the calf was strong and normal in every respect, and remained so.

Conclusions

Sulfapyridine, in moderate and reducing doses, appears to be a valuable *addition* to the therapy of calf septicemia. Occasional toxic effects can be overcome by the use of small doses of sodium bicarbonate. Its use must be accompanied by sanitation, reduced diet, intestinal antiseptics, and any other symptomatic treatment that is indicated. Transfusion of defibrinated dam's blood is of great value in maintaining the strength of the patient.

Butler, Pa. DANIEL P. EHLERS.

* * * *

Errata

Page 308 (May issue), last sentence in first column should read: "Tablets containing 4gr of pepsin, 1gr of pancreatin, and ¼gr of diastase. . ."

Page 344, this issue, in second paragraph of second column, instead of $25 read $250.

Treatment of Bovine Mastitis with Sulfanilamide

Considerable data have been published on the use of sulfanilamide in the treatment of bovine mastitis, but to our knowledge, none of these reports has dealt with a Group C, Beta hemolytic streptococcus infection.

According to Gildow, Fourt, and Shaw,[1] sulfanilamide produced poisoning in one to three days when the total daily dose was 15gm per 100 pounds body weight, while five grams per 100 pounds body weight had little or no detrimental effect on nine of 11 cows treated. They stated that blood and milk levels in cattle were maintained over a period of 12 hours either following an initial dose or after the last dose of a period of treatment. This insures a reasonably constant level of sulfanilamide in the blood of cows that are dosed twice daily at 12-hour intervals. In the experiment described in this paper, an attempt was made to maintain a maximum blood concentration of sulfanilamide for a reasonable length of time to study the effect of this drug on the elimination of Beta hemolytic streptococci of Lancefield's Group C.

History

A nine-year-old Jersey cow weighing approximately 900 pounds was brought to the veterinary clinic of Kansas State College with a clinical diagnosis of chronic mastitis in the right front quarter. The milk flow from this quarter was scant and watery, and palpation of the quarter disclosed a great deal of induration and partial atrophy. No other clinical symptoms were present.

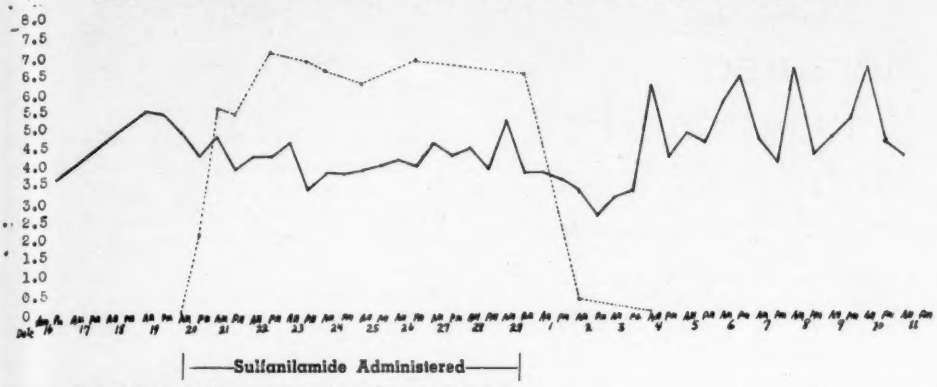
The case was brought to the author's attention by the physician attending the family of the cow's owner. This physician had treated the family of five for septic sore throat some four months previously, advising them at that time to discontinue the consumption of milk from the cow. They had recovered following the administration of sulfanilamide, but suffered a

[1] Gildow, E. M., D. L. Fourt and A. O. Shaw, 1938. Sulfanilamide in the Treatment of Streptococcic Mastitis. *Jour. of Dairy Sci.* 21:12, pp. 759-766.

relapse less than four months later. The physician learned that milk from this cow was again being used by the family. Milk

The highest concentration of sulfanilamide reached in the blood was 6.9mg% on the fourth day of administration. The level

CHART I. SULFANILAMIDE CONCENTRATION AND BETA-HEMOLYTIC STREPTOCOCCI COUNTS.



samples were found to contain Beta hemolytic Lancefield Group C streptococci, and the animal was brought to the clinic for this experimental study.

Procedure

Sulfanilamide was administered in gelatin capsules in 30gm doses. The first day, three doses were given at eight-hour intervals, followed by two daily doses of 30gm each at 12-hour intervals for the following nine days. Milk and blood samples were taken at intervals to determine the concentration of sulfanilamide.

Milk samples were collected aseptically twice daily, beginning three days previous to, and continuing to the eleventh day following administration of the drug. They were plated in blood agar, and total counts of Beta hemolytic colonies were made from the blood agar plates.

Results

This animal lost approximately 200 pounds during the experiment and showed an occasional slight inappetence. No abnormal rise of temperature was noted throughout the treatment. The milk flow was decreased about one third, but became normal in physical properties.

of sulfanilamide in the milk paralleled that in the blood throughout.

The streptococci, found only in the right front quarter, were present in all the samples from this quarter. The sulfanilamide level in the blood and the numbers of streptococci present are shown graphically in Chart I.

Summary

A cow affected with mastitis due to a Beta hemolytic Lancefield Group C streptococcus was treated with sulfanilamide for 10 days. The blood level of sulfanilamide was maintained between 6 and 7mg per 100cc for at least eight days. Bacterial analysis of the milk samples taken twice daily during administration of the drug and for 11 days afterward revealed the continued presence of the causative organism. Some decrease in the number of streptococci in the milk was evident during the period of highest concentration of the drug in the blood stream and in the milk, but this reduction was only temporary.

RUSSELL A. LEEPER[2], K.S.C. '40.
Manhattan, Kans.

[2] The author is indebted to Professor V. D. Foltz and to Miss Alice Kimball for valuable advice and technical assistance.

Oxygen-Foam Therapy in cases of Retained Placenta and Metritis

By E. G. BAXTER, Webster, New York

THE placenta which remains in the uterus after parturition affords a constant source of infection, making it easy for streptococci and other pathogenic microorganisms to penetrate into the placenta materna. The general opinion is, therefore, that it is advisable to remove the after-birth as soon as is deemed safe from a surgical standpoint. Although there is a diversity of opinion as to when such removal is desirable, authorities agree that the eighth day is about the limit. The author's experience, over a period of 20 years, leads to the belief that conditions are most favorable 18 to 36 hours after parturition.

The uterus is very large immediately after delivery and for three days following. Lack of tone of the muscles and blood vessels, or infections, delay proper drainage of blood from the cotyledons, stopping natural shrinkage and causing the chorionic tufts to remain anchored tightly. This condition may be brought on by a long or difficult delivery with attendant depression of the animal, improper feeding, unfavorable barn conditions or infections such as Bang's disease. Each succeeding day the uterus decreases in size, but after 24 hours a secondary infection from without invades the cotyledons, inflaming them and keeping them enlarged. It may happen that the placenta is easily detached from the cotyledons in the neighborhood of the cervix, but the farther one penetrates into the horn, the more the horn bends and it is difficult to follow into these bends with the arm. The use of force may cause wounds which open the doors to bacterial invasion. Retentions are especially frequent after *Br.abortus* infections. Removal of the placenta at this time cannot be accomplished with safety unless some method is used to control the bacterial content and to protect the exposed surfaces of the cotyledons.

The cervix begins to shrink immediately after calving and in most cases is too small to admit the hand after 72 hours. If unattended, the cervix may lose its muscular tone in seven or eight days, allowing the decomposed placenta to be expelled or removed in part or whole. However, to permit the placenta to remain in the uterus after 24 hours with no attempt to control bacterial growth invites metritis and endometritis with the possible consequence of sterility.

■ Modern uterine therapy makes it imperative that some method be used which will control bacterial growth, remove the toxins and shrink the cotyledons. A method which does this, and also assists in mechanical removal of the placenta, is of untold value to the veterinarian in protecting the health and future breeding value of the animal, not to mention elimination of the personal danger and the very disagreeable character and odor of this work.

A novel method of combining chemical and physical means was first described by Detlefsen[1] who found that foams containing nascent oxygen not only had a definite bactericidal action, but also greatly helped to loosen the placental membranes from the cotyledons. Werner[2] reported 54 cases in which this principle was very successful in all but four.

A similar preparation, utabs,[3] which combine the nascent oxygen foam principles with the deodorizing properties of osmotically purified kaolin and the lasting germicidal properties of 3 -chloro - 4 - hydroxy-diphenyl was used in over 100 cases of retained placenta and metritis by the author. When it was compared with the material employed by the foreign investi-

[1] Detlefsen, 1936. *Tierartzl. Rdsch.*, 50, p. 909.
[2] Werner, 1938. Inaugural Dissertation, University of Leipzig.
[3] Furnished through the courtesy of the R. J. Strasenburgh Company, Rochester, N. Y.

gators, it was found that the kaolin, in addition to its deodorizing properties, helped to eliminate mechanically small pieces of tissue, coagulated blood, etc.

■ The technique used is as follows:

Temperature readings are used to determine whether immediate removal should be undertaken. If the temperature is normal, a preliminary exploration of the uterus and horns is made to estimate their condition and the amount of inflammation in the caruncles. The placenta is then retracted with some force to allow the placing of eight to ten broken utabs between it and the uterus. Five to 30 minutes are allowed for the utabs to become actively foaming, after which removal of the afterbirth is accomplished in the usual way.

While replacing the tips of the uterus to their full length, six to ten utabs are placed about midway to the end so that the germicidal and oxidizing action may be continued.

The greater ease of removal, the oxidizing of the separated surfaces, the decidedly favorable impression on the owner, and the elimination of the disagreeable odor are all highly desirable features.

If the temperature is 103°F. or over, some metritis has already developed and immediate removal is contraindicated. In these cases 12 to 16 utabs are placed well into the horns in various places and one in the vagina to stimulate straining. Three hundred grains of sulfanilamide are given orally. An examination in 12 hours usually finds conditions greatly improved and the placenta can, as a rule, be removed at this time. If not, four to six utabs are placed in the uterus daily until removal can be accomplished. The action of the utabs, extending to all parts of the uterus and horns, reduces the danger of infection to both the animal and the attendant.

Case Records

Case 13, M. M., Ontario, N. Y.—Purebred Holstein, five years old. Freshened April 16, 1938; veterinarian called April 18.

The temperature was 103°F. The cotyledons were inflamed and the placenta was not removable. Twelve utabs were placed in the horns and 300 grains of sulfanilamide were administered. The next day the temperature was 102°F., and the inflammation was greatly reduced. Six utabs were placed in the horns and the placenta was removed. Four utabs were left in the uterus to keep down infection and to act as a cleansing agent. The animal was successfully bred at a later date.

Whether or when to remove a retained afterbirth and measures to circumvent infection are perennial problems confronting every dairy practitioner. This discussion gives one practitioner's solution.

Case 18, I. C. D., Ontario, N. Y.—Grade, four years old. Freshened April 25, 1938. Veterinarian called April 26. Temperature 102°F. On examination, the placenta was found firmly attached, but in good condition. Six utabs were broken in various parts of uterus. In about five minutes considerable foaming action had taken place and the placenta was easily removed. There was no hemorrhage, and but little odor. Six whole utabs were left in the uterus.

Case 37, J. M., Webster, N. Y.—Purebred Holstein, six years old. Freshened September 13, 1938. Veterinarian called September 15.

Temperature 102°F. The placenta was firmly attached. Ten utabs were broken in various parts of the horns and after five minutes removal was started. The placental tufts felt clean and firm, and there was no hemorrhage. Six utabs were left in the uterus.

Case 47, S. A., E. Rochester, N. Y.—Grade, about three years old and in very poor condition. The barn was poorly built and open, so that manure on the floor was frozen. This cow aborted a seven-months calf December 1, 1938. Veterinarian called December 3.

Temperature 103°F. The placenta was badly decomposed, with a strong odor. Because of the low value of the animal and other circumstances, it was decided to do as much as possible in one call. Six utabs were broken in the horns, and after 15 minutes as much of the placenta was removed as could be taken without injuring the cotyledons. Six more utabs were placed in the uterus and 300 grains of sulfanilamide administered. The owner reported that the rest of the placenta came away in a few days, and that the animal appeared normal.

Case 76, H. H., Webster, N. Y.—Percheron mare, seven years old. Dropped colt May 16, 1938. Veterinarian called May 17.

The retained placenta was removed fairly easily. Eight whole utabs were placed in the

uterus and 240 grains of sulfanilamide were given. No further treatment was necessary.

Cases 24, 25 and 26, N. R. P., Webster, N. Y.—Three purebred Holsteins from a Bang's disease-free herd freshened on July 11 and 12, 1938; all had normal calves. Veterinarian called July 13. The temperatures were all normal. Ten utabs were broken into each uterus and from five to 30 minutes allowed for them to start their action. Two placentas came away perfectly and the third nearly so, except for a small portion in the horn tip. Six whole utabs were left in each uterus. All three were successfully bred in the normal time and on the first breeding.

Case 94, F. M. W., Ontario, N. Y.—Purebred Holstein, four years old, which had dropped one live calf and later a large dead calf on August 11, 1938. The live calf was normal and its placenta came away without help. The herd has been Bang's disease free over a long period.

Temperature 106.5°F. No appetite. The uterus and placenta were in very poor condition with great amount of odor on August 12 when examined. Twelve whole utabs were placed in the uterus, but no effort was made to remove the placenta. Three hundred grains of sulfanilamide and stimulants were administered.. *Aug. 13th:* temperature 106°F. and general condition not improved. Ten utabs were placed in the uterus and the sulfanilamide and stimulants repeated. *Aug. 14th:* temperature 105°F., considerable discharge. Six utabs were placed in the uterus and most of the placenta was removed. Four whole utabs were left in the uterus; 180 grains of sulfanilamide and stimulants were given. *Aug. 15th:* temperature 104°F. and little appetite. Eight whole utabs were placed in the uterus. The stimulants were continued, and forced feeding of gruel was started. *Aug. 16th:* temperature 104°F., the general condition showing little change. Four whole utabs were placed in the uterus and the other treatment repeated. *Aug. 17th:* temperature 103.5°. The animal was eating some hay and fresh corn stalks, and the condition of the uterus was greatly improved. Two utabs were inserted in the vagina to induce expulsion of slight discharge and the stimulants were continued. *Aug. 18th:* temperature 102.5° F., appetite better. One utab was placed in the vagina and stimulants were given. *Aug. 19th:* temperature 102.5°F., appetite fair. The cow was in very poor flesh, and was sent to another farm for pasturing; no further treatment was given. *Sept. 16:* examination showed the uterus to be nearly normal, with no discharge. The animal was eating well. *Feb. 5th, 1940:* in good condition and carrying calf.

Sulfanilamide Treatment of Acute Mastitis in Dairy Cattle

After many trials and failures with various mastitis treatments, including udder injections, formalin, turpentine, saline purgatives, foreign proteins, boiled milk, and numerous other remedies, the following group of treatments have been found most effective:

1. Immediate reduction of protein intake, with reduction of grain to 14% protein, or less, and not forcing the animal for remainder of the lactation period.

2. If in the first 24 hours, cold or ice pack the udder every two hours. If after 24 hours, use hot packs.

3. Milk the affected quarters hourly if possible; at least every two hours.

4. Sulfanilamide powder, 400 to 600 grains divided into three doses, each 24 hours for two days. If advisable to adminster the drug longer, reduce the dose to one-half the amount. Sometimes acriflavine (1gm in 500cc sterile water, administered intravenously) is beneficial. (Milk is colored for several days after acriflavine.)

5. Mastitis mixed bacterin: 10 to 15cc first dose; 15 to 25cc second dose on following day.

6. Massage udder with creamy white liniment after acute heat and pain recede. Doctor Baker's formula[1] has been very satisfactory and is inexpensive to make.

In our experience, more animals have returned to successful production under the above treatment and remain negative to the brom-thymol test longer than under any other method of treatment that we have tried.

H. B. WALTERS.

Newark, N. Y.

[1] Into a gallon bottle pour one pint of lukewarm water containing two ounces of stronger ammonia. Add another pint of warm water to which has been added two ounces oleic acid, and shake the bottle well. In a pint graduate dissolve four ounces ammonium chloride in warm water, and pour this into the bottle, again shaking well. Add a dram of red aqueous color. Pour four ounces of methyl salicylate into the pint graduate and add 12 ounces of turpentine, colored pink with red oil color. Pour this into the bottle, adding enough warm water to fill it. Shake well, and set in a warm place such as the kitchen for a week to "ripen." This makes a creamy pinkish-colored liniment with a pleasing odor. It can be used for many other conditions, such as bruises, sprains, shoulder galls, etc.

Zephiran in the Treatment of Foot-rot in Cattle

By J. W. SCALES, Blacksburg, Virginia
Department of Biology,
Virginia Polytechnic Institute

DURING the past several years a dozen or more cases of foot-rot have occurred annually in a herd under the writer's observation. Various remedies have been used in treating these cases, with recovery very slow. This year the writer has been using zephiran.*

Zephiran is a trade name for a mixture of high molecular alkyl-benzyl-ammonium-chlorides derived from the fatty acids of coconut oil. It is an efficient antiseptic of high germicidal and bacteriostatic potency, stainless, of pleasant odor and, in proper dilutions, non-poisonous and non-irritating to tissues.

The specific cause of foot-rot is probably a filterable virus but *Actinomyces necrophorus* is an important invader and mainly responsible for the lesions. Both are found in soil and filth. These organisms also produce foot-rot in sheep and reindeer, sore mouth in sheep, necrotic stomatitis in calves and bull nose in pigs.

The first evidence of an attack of foot-rot to attract the attention of the herdsman is a slight lameness which becomes more marked rapidly. Previous to this there occurs a moist area just above the horny part of the cleft of the foot which gradually reddens and assumes a feverish, inflamed appearance. It first becomes visible either at the front or at the back part of the cleft, more often the latter. The inflammation penetrates rapidly beneath the horny tissue, resulting in an ulcerous opening which exudes a thin purulent fluid. The lameness increases, and the region of the foot above the hoof becomes swollen and warm to the touch. The exudate from the erosions has a disagreeable odor.

The erosion progresses, if no treatment is applied, with rapid formation of fistulous passages beneath the horny covering of the foot. Along with this the softer tissues of the interdigital space gradually become necrotic and purulent. The invading organism possesses marked burrowing propensities, and the result of its invasion is that large areas of the hoof become loosened from the sensitive tissues lying beneath.

When the loosened horny tissue is cut away, it is found that the undermining process has advanced until numerous ulcerative channels have been formed which are filled with grayish purulent matter, and that the encroachment upon the healthy area is persistently and constantly being extended. One or both claws of the foot may be affected.

The hoof of a cow suffering from a chronic case of foot-rot grows out rapidly and becomes very hard. The toes are often so thickened and lengthened that the front part of the foot is raised above its natural incline and the tendons at the heel are subjected to additional strain, all of which tends to increase the lameness and awkwardness of the victim. These thickened and elongated toes frequently attain an added length of three or four inches, causing them to curl up.

The cow becomes so lame when this disease has become thoroughly established, that she will not walk unless forced to do so. Her appetite is seriously impaired, and she loses condition and weight rapidly, with a corresponding diminution of the milk flow.

One of the first steps to be taken in the treatment of a cow affected with foot-rot is to cleanse the foot thoroughly by scrubbing with a stiff brush and water. The infected cavity is then opened and all diseased tissue removed, leaving a clean open wound. After this has been accomplished, zephiran is applied to the affected parts, care being taken to ensure that it reaches all the diseased areas. Often no further

* Manufactured by Winthrop Chemical Company, New York, N. Y.

treatment is necessary in mild cases or even in extensive cases if the affected surfaces are exposed to the action of this solution.

During the past spring eight heifers on pasture were treated for foot-rot. These heifers were all in the early stages, and, after a thorough application of zephiran, no further treatment was necessary. However, with six cases occurring in the milking herd, an average of three applications was necessary. This, no doubt, was due to their walking through littered lanes, thereby reinfecting the wounds.

One advanced case required packing with zephiran because of extensive invasion and decay of the soft tissues, and several such treatments were necessary before recovery occurred.

Conclusions

Zephiran has proved effective in the treatment of foot-rot in cattle, most cases recovering after only one application. It has proved far superior to the remedies previously used for this condition. It merits a trial in other necrophorus infections.

Iliac Thrombosis

A mare recently shipped broke out in cold sweat suddenly in the morning as reported by the attendant. Within 20 or 30 minutes she went down and after going down seemed normal, eating and drinking and showing no pain. I was called early in the night. I found her down, but otherwise all right. She would get to her feet, but after being on her feet about five minutes she would show extreme pain in the hind quarters. If allowed to lie down she became normal again. If kept on her feet she would tremble, place hind feet forward, sweat and go down. After lying five minutes she would seem normal. As I am a 1938-model veterinarian, and as the mare was valuable, I called in another, more experienced veterinarian for consultation. I had given a tentative diagnosis of founder hurriedly at the beginning, even though the feet were not hot. The older veterinarian gave a diagnosis of sciatica and prescribed salicylates with directions to allow the mare to remain lying with intermittent standing. She began improving slowly within four or five days. At the end of a week she was much better, but still not well. I will appreciate comment on diagnosis and treatment.—*C. S.*

Comment: For diagnosis, see the title. Treatment: rest. See VETERINARY MEDICINE, Vol. 34, No. 8 (August, 1939), p. 487.

Tear in the Uterine Wall in a Mare

On page 290 of the May issue of VETERINARY MEDICINE under the heading "Septic Metritis" there appears a very interesting case report, with comments by two veterinarians, neither of whom made a diagnosis.

In describing the case, the veterinarian in charge does not state whether or not he made a careful examination of the uterus to find out whether or not it was torn. From the fact that he does not state that he made such an examination, I am led to believe that none was made; if it was made, he may have missed the tear in the uterus which caused the death of this mare. In all probability the tear was low enough so that there was considerable drainage from the uterus into the abdominal cavity. I have seen a number of mares die in exactly the same way as he describes in his case report, and in each case there has been a rupture of the uterus. The contraction of the uterus and the expulsive efforts of giving birth to a foal are very powerful, and many times mares injure themselves prior to the time that the veterinarian arrives. Many colts are born alive and the dams die a short time afterward, and not always from hemorrhages. I am unable to explain why a mare with an opening in the uterus may die so quickly, while a cow may live for several days and even sometimes recover, except on the grounds of shock.

The comments by "F. H." are good. His reasoning that this animal could not have died from septic metritis is sound. I also agree with him that no treatment would have been effective. However, blood transfusions, glucose and physiological salt solution and chemotherapy intravenously might have prolonged the life of this animal, but my opinion is that it would not have saved her.—*T. O. B.*

Ten Achievements of Veterinary Medicine

1. Veterinary medicine assures a food supply for the people of nations having a veterinary service. Backward nations not maintaining a veterinary service suffer from periodical famines due to animal plagues. In countries having a good veterinary service famines do not occur except as a result of devastation by armed forces.

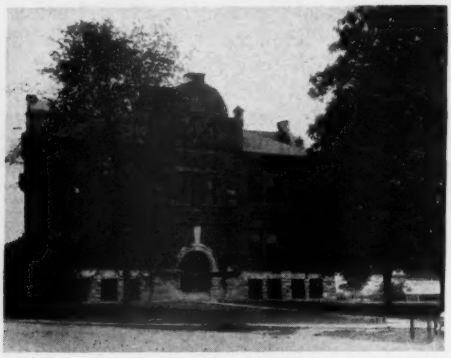
2. Throughout the Dark Ages and the Middle Ages, recurrent famines followed by pestilences decimated the populations of all nations of Europe. These were invariably preceded by animal plagues that destroyed the husbandman's animals and thus his means of transportation and of tilling the soil—that is, of producing food for the people. These devastating animal plagues continued right down to the time (nearly 200 years ago) that a scientific veterinary service was established and brought them under control. This has enabled the population of Europe to quadruple and still not suffer the famines of old.

3. It is this control of animal plagues that makes possible our present agriculture; that has enabled our live stock industry to make the vast improvements in breeds of live stock that the last half century has witnessed. Certainly the productiveness of beef cattle, dairy cattle, swine, and poultry has doubled in that period due to the interchange of breeding animals. This free exchange would not have been possible without control of animal diseases exercised by our veterinary service.

4. Research by veterinarians has contributed immensely to the wealth of the nation. It has developed an almost perfect preventive for hog cholera, which formerly swept great areas of our agricultural districts, destroying up to 90% of the swine in some districts. An Army veterinarian developed a rinderpost vaccine that alone makes possible the present agriculture of the Philippine Islands. Veterinarians developed a successful treatment for milk fever, which a generation ago killed 90% of the cows it attacked—and it attacked the high producers in every good dairy herd, not only entailing immense loss to the dairymen and depleting the milk supply, but effectually preventing improvement of the breed by removing always the best animals. They have developed a fairly successful vaccine for the prevention of distemper in dogs, the most prevalent disease of that animal, a measurably effective preventive for rabies of dogs, and successful measures for preventing or combating all but one of the important infectious diseases of poultry. More recently veterinarians developed a nearly 100% successful means of preventing encephalomyelitis, or sleeping sickness, in horses. This by no means exhausts the list of veterinary research achievements, but it is indicative of the incalculable value such achievements have had for all the people.

5. The ancient plagues of animals which in the long ago starved whole populations and destroyed nations still exist, ever ready to spread devastation as of yore, but are held in check by our veterinary service. Foot-and-mouth disease of cattle, the most infectious disease known in man or animals, has repeatedly invaded our country, and as frequently been eradicated; sometimes at a cost of millions of dollars, it is true, but at no time has the cost of eradication been 1% of the yearly cost of the disease were it permitted to extend over the whole country. In the past 20 years, veterinarians have practically eradicated cattle tuberculosis from the whole country, although it was so widespread when this work began that it cost our cattle industry upwards of a hundred million dollars annually. Tick fever of cattle was formerly spread over 15 of our southern states, and frequently invaded territory as far north as Pennsylvania, Illinois, and Nebraska. Like tuberculosis, the loss from it exceeded 100 million dollars a year. As the result of a campaign against it begun by veterinarians in 1906, it has been eradicated from all but a few counties in Florida, where it is perpetuated by wild deer that live in impenetrable swamps and are protected by

game laws; and a few counties in Texas along the Rio Grande, where it is reintroduced from time to time by cattle from Mexico that swim across the river. Contagious pleuropneumonia of cattle was once



Bacteriology Building and office of the Dean, Division of Veterinary Science, Michigan State College.

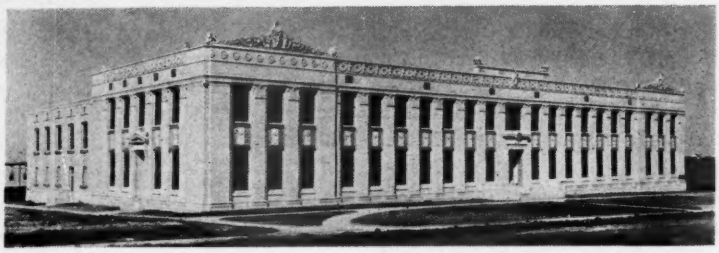
prevalent in all the country from Illinois east. On account of this disease, the countries of Europe refused to permit the entrance of American cattle, and our export cattle business was on the way to being completely destroyed. The disease was eradicated by veterinarians and has ever since been kept out, and now there is no country in the world that bars American cattle.

6. Veterinary research has contributed beyond measure to the control of disease in man. It was in the study of tick fever in cattle that it was discovered that insects may carry certain diseases. It was this basic contribution of veterinary medicine that led to the discovery by Maj. Walter Reed, an Army surgeon, that yellow fever is spread by mosquitoes. This released large areas of the world from a plague that had struck terror into many cities and almost depopulated them. But for this discovery the Panama Canal, for example, could not have been constructed; Havana would not be now visited by American tourists, but would be the pesthole it had been for more than 300 years; and New Orleans and every other tropical and subtropical city would be in constant danger. Present transportation facilities all over the world could not be enjoyed—could not exist. Malaria control all over the world owes its existence to this basic veterinary discovery, and hundreds of millions of people have been saved from a lingering death or a long, costly, debilitating illness as a consequence of it.

7. The discovery of chick embryo vaccine for sleeping sickness in horses may possibly result in the development of a preventive for a number of similar nerve and brain diseases in man, including sleeping sickness and infantile paralysis, and there is some promise that a means for preventing influenza in man may grow out of work veterinarians are prosecuting at the present for the control of an almost exactly similar disease in swine. In fact, there are about 70 different diseases and parasitisms of animals that are communicable to man. For a number of these, animals constitute the natural pool or reservoir, and there is little prospect of ever protecting man from

Veterinary Clinic and Department of Pathology, Ohio State University.

them except as we are able to control them in animals, which of course is the veterinarian's job. Tuberculosis of cattle is an example; as a result of eradicating this disease from cattle, veterinarians are preventing the deaths of thousands of persons in this country every year from the bovine type of tuberculosis, and the sickness of tens of thousands annually. That type of tuberculosis was formerly common in this country, particularly among children, and it occurs still in every country in the world where cattle tuberculosis prevails. It was responsible for most of the cases of hip-joint disease in children, as well as hunch back, swollen glands in the neck, and scrofula. It has been an appreciable factor in reducing the death rate from tuberculosis in this country from over 200 to less than 50 per hundred thousand of our population.



Veterinary Building, Texas A. and M. College, College Station, Texas.

8. Veterinary inspection is responsible for the fact that the people of the United States enjoy the best and the most wholesome food of animal origin of any nation in the world. I refer to meat, meat products, milk, cream, butter, cheese, ice cream, fish, etc. Veterinarians in the employ of the federal government inspect more than 75,000,000 animals annually at the time of slaughter at our packing houses and remove from the food supply more than 300,000 carcasses of animals that are diseased or otherwise unfit for food. In addition many times this number of parts of carcasses are condemned and destroyed.

Unfortunately only about two-thirds of the animals slaughtered for food are processed in plants having veterinary inspection. About one-third are slaughtered in local plants—that is, establishments that do not do an interstate business. Only a very small minority of such plants have adequate inspection. An extension of our veterinary service to include these also is badly needed, but will have to be provided by the states. The federal government cannot reach concerns doing business wholly within a state.

9. The veterinary corps of the Army inspects and certifies to the wholesomeness of all food of animal origin used by the Army, and civilian veterinarians under the supervision of Army veterinarians inspect all foods of animal origin used by the CCC throughout the country. The government is saved an immense sum by this inspection, which eliminates hundreds of thousands of pounds of unfit or substandard food products.

10. In addition to the veterinarians engaged in rendering agriculture and the whole public the services mentioned in the foregoing, there are eight or nine thousand engaged in private practice, restoring ailing animals to health and preventing the loss to their owners the death of these animals would represent. Their work extends to practically every farm where live stock is raised and homes where pet animals are kept. They too have a large share in disease prevention by the use of serums and vaccines, and perhaps still more by the better sanitation that they teach the owners to observe in the care of their animals. No other humane force—not even public sentiment—relieves or prevents so much suffering among animals as the work of these men, who are on duty throughout the land, trained and experienced in animal disease control.

Recent Materia Medica

III. Vitamin K.

By PROF. VICTOR LEWITUS
Nutley, New Jersey

Historical Sketch

One of the most recent additions to the ever-increasing members of the vitamin family is vitamin K. The existence of this fat-soluble vitamin was first suspected some 10 years ago (1929), when Dam of Copenhagen experimented with chickens. In studying certain phases of lipoidal metabolism, he placed newly-hatched chicks on a fat-free diet. Several weeks later, the chicks developed hemorrhages into the skin, mucous membranes and certain other tissues. Definite progress in the recognition of a specific factor did not occur until 1935, when Dam and Schönheyder determined that the essential factor without which the hemorrhagic tendency occurred, was present in certain natural food products and was unlike either vitamin A, vitamin D, or vitamin E. Since Dam referred to this vital element in the animal diet as the "Koagulation-vitamin," he selected the first letter as the abbreviation; hence the designation vitamin K. The work of Holst and Halbrook in California (1933) must not be overlooked, for they observed the curative effect of feeding fresh cabbage in the nutritional disease of chicks characterized by intramuscular, subcutaneous and intra-abdominal bleeding. Furthermore, Halbrook noticed that such diatheses could be prevented by supplying an extractive of alfalfa or putrefied fish meal—later determined to be excellent sources of vitamin K. In 1937, Almquist reported improvement in the process of concentrating the anti-hemorrhagic vitamin, obtaining a crystalline substance with a low melting point from certain hydrocarbon extractives. Although the prophylaxis and cure of the hemorrhagic diathesis in chickens stimulated some degree of interest, it was not until some time later, when other animals as well as humans were found to react favorably to the effects of feeding the vitamin, that genuine interest was manifested. Vitamin K has proved its worth in the mammal in certain diseases of the biliary tract, and its application is being widened to include other disorders; for example, those occurring in the intestinal canal. Undoubtedly, more uses for the vitamin will be found as it becomes more readily available.

Sources

Vitamin K is present in relatively large amounts in certain green, leafy vegetable or plant products such as alfalfa, kale, and spinach. It is also found in soy bean meal, cauliflower, dry carrot tops, and hemp-seed, as well as in the fatty portion of hog's liver and egg yolk. Putrefied fish meal, such as putrefied sardine meal, and the isolate obtained from alfalfa are the richest of the present natural sources. The other sources, derivatives or synthetic materials, are discussed below.

Chemistry and Properties

Since more careful observations may be made with pure crystalline material, and this form also permits parenteral therapy, much effort has been directed toward the isolation, purification and synthesis of this very complex vitamin. As already mentioned, alfalfa and putrefied fish meal have been the best commercial sources of the substance. At the St. Louis University, McKee and others have isolated vitamin K_1, a light yellow, oily substance obtained from alfalfa, which when cooled forms a crystalline solid. Almost simultaneously, Dam and other collaborators isolated a similar preparation. From putrefied sardine meal, the St. Louis workers also isolated vitamin K_2, a light yellow, crystalline solid melting at 50° to 52° C. Both vitamin K_1 and vitamin K_2 are sensitive to light and alkaline substances, and readily absorb hydrogen, yielding colorless substances which are quickly oxidized in the presence of air. The original work of Hooker, Steyermark (1936), and others in synthesizing naphthoquinone derivatives such as lapachol and lomatiol prepared the

way for the synthesis of the antihemorrhagic vitamin. Almquist and Klose have found recently (1939) that pure synthetic phthiocol (also isolated from tubercle bacilli) is one of the simpler compounds of a series that improves blood-clotting time. Also at this time, McKee and others announced that vitamin K_1 is a quinone, or more exactly a naphthoquinone, having the empirical formula $C_{31}H_{46}O_2$. The constitution of vitamin K_2 is somewhat dissimilar. The synthesis of vitamin K, the pharmacological activity of which is apparently identical with that of natural vitamin K_1, has just been accomplished by Almquist and Klose, and also by other workers. Summarizing the important properties of vitamin K, it may be stated that it is fat-soluble, sensitive to light, readily affected by alkalies, and non-saponifiable. At present the product is chiefly marketed in the form of a peanut-oil solution. Toxicity has not been reported thus far.

Assay or Standardization

In most of the assay work involving vitamin K determinations, the prolongation of clotting time is selected as a convenient and reliable basis of estimating potencies. There are, in general, two methods of making an estimation of activity, the prophylactic and the curative. Dam and Schönheyder favor the curative method. Young chicks are placed on a basal diet containing ample food elements except vitamin K. The animals are maintained on this diet until disease develops with a marked delay in the clotting of the blood. The animals are now divided into several groups, and each group is fed the substance to be tested in various quantities. The minimum amount per gram body weight, which within a certain period (three days) is capable of restoring the rate of clotting to normal, is called a unit. The prophylactic (Almquist-Stokstad) method of estimating potency is probably more popular. Young chicks are supplied with a vitamin K-free diet, supplemented with vitamin K in definite proportions. The smallest quantity per gram body weight that will prevent prolongation of the clotting time beyond normal is called a unit. One unit of the Almquist-Stokstad prophylactic method is approximately equivalent to 25 Dam-Schönheyder curative units; in other words, the Almquist unit is the weightier of the two; but it is best not to attempt to translate one unit in terms of the other, since the criteria of measurement are too dissimilar.

Function of the Vitamin

It has already been pointed out that the early work of Dam with chickens on a fat-free diet, with subsequent hemorrhages under the skin, mucous membranes, etc., led this worker to suspect the existence of vitamin K. Almquist and Stockstad furnished evidence that vitamin K can be produced as a result of bacterial action in the animal body, and that it is usually present in the lower part of the intestine even when animals are maintained on diets lacking the vitamin itself. It has been found that absorption from the lower part of the intestine is at a minimum in chicks, but in mammals occurs readily and for this reason vitamin K deficiency is rare except in pathological conditions in which there is interference with intestinal absorption. Hemorrhagic disease due to shortage of vitamin K has been observed in many animals. It has been induced in chickens, ducklings, goslings, pigeons and canaries—in mild form in the latter two. It is not surprising that the hemorrhagic diathesis cannot be produced in the normal rat, guinea-pig, dog or man, for as has been pointed out, mammals obtain vitamin K not only from the diet but also as a result of bacterial activity within the intestine and can develop a deficiency only when absorption of the compound from the intestine is interrupted. It has been suspected but not proved that some of the bloody dysentery of swine, so common following severe drouths, is due to vitamin K deficiency.

Only recently has vitamin K deficiency in higher animals been explained on a more rational basis than before. It has been observed that faulty absorption occurs when bile is excluded from the intestine. The biliary secretion has long been known to aid in the absorption of fats. Since vitamin K

is a compound that is fat-soluble, it is only logical to suspect that in the absence of bile secretion or bile salts the vitamin cannot be absorbed.

It is well recognized that clotting is one of the most striking properties of blood, but there is little agreement among physiologists as to its mechanism. Available evidence indicates that clotting apparently involves two consecutive reactions: first, interaction between the prothrombin within the plasma, the calcium, and the platelets (or tissue extract) to form thrombin; second, reaction between the thrombin and the fibrinogen to form fibrin. Attempts at further analysis of the phenomenon lead one into a maze of contradictory theories. Rats, according to Greaves and Schmidt, develop a prothrombin deficiency in the presence of biliary fistula, and Hawkins and Brinkhous have observed the same condition in dogs. The bleeding tendency which may develop in humans having obstructive jaundice has long been recognized. The explanation of the condition is a source of great controversy, since it has been an accepted belief that the resorption of bile is without direct effect on the coagulation of blood. The concentration of bilirubin, bile salts or cholesterol in the blood in jaundice bears no relation to the disturbance in coagulability. The level of blood calcium, if low, ordinarily does not determine the bleeding tendency; and the amount of fibrinogen is also rarely diminished in jaundiced patients. The presence of antithrombin or antiprothrombin has been postulated, but this has not been confirmed. Furthermore, Howell has suggested the possibility that heparin is responsible for normal blood fluidity, but this seems untenable since evidence points to no increase in the heparin concentration of blood in cases of jaundice. The most recent investigations seem to point to the fact that decreased prothrombin content of the plasma accounts for the hemorrhagic tendency in obstructive jaundice. It is very important to observe, in passing, that slight reduction of the prothrombin level has little effect on blood coagulation. The coagulation time, as measured by accepted methods, shows very little delay until over 80% of the prothrombin has been lost. Reduction below this critical level, however, is accompanied by marked disturbance of the clotting mechanism. This observation explains why humans having prothrombin deficiency previous to an operation show no tendency to hemorrhage, but when the prothrombin level is further reduced as a result of operative trauma, the tendency to bleed may become manifest and death due to blood loss ensue. Fortunately, the converse is also true; that is, only a slight increase in prothrombin level often suffices to control the tendency to hemorrhage, and this partly explains the value of transfusions in certain instances. Smith, Warner and Brinkhous have recently shown that animals suffering injury to the liver from chloroform anesthesia or carbon tetrachloride poisoning tend to bleed because of lowered prothrombin content; but other causes may contribute to the condition also. Roderick and Quick have noted

It has been suspected, but not proved, that some of the bloody dysentery of swine, common following severe drouths, is due to vitamin K deficiency

that animals fed spoiled sweet clover hay suffer from prothrombin deficiency which can be cured by transfusion and feeding alfalfa. Since in cases of obstructive jaundice (in humans) and biliary fistulas (in lower animals) the intestine receives little or no bile, the absorption of vitamin K is interrupted. After a period of time, the tendency to bleed then develops since the prothrombin level falls far below normal. The indicated treatment consists of bile or vitamin K, or (more recently) both vitamin K and bile salts, for the latter renders the vitamin K effective because of the absorption that follows.

Practical Application

It is too early to determine all the possible uses of vitamin K in veterinary practice. It suffices to state that, in general, the vitamin has a place in the therapy of any condition in which blood prothrombin is lowered because of shortage of the antihemorrhagic substance. Such deficiencies may be expected in disorders which shut off the supply of bile to the intestine or decrease production of the bile salts. Cases of acute or chronic liver atrophy, obstructive jaundice due to lithiasis, partial or complete biliary fistula, or anatomical obstruction of the biliary passages due to neoplastic processes, all call for administration of vitamin K and bile salts—the combination furnishing more prompt results than either remedy alone. Although the degree of vitamin K deficiency tends to be greater in long-lasting cases, it has been observed that obstructive conditions of only several weeks' duration cause marked deficiency of the vitamin. Various tests for determining clotting time are available, particularly the one by Quick, but these require modification to render them practical.

Present-day methods of determining vitamin K deficiency are based on the determination of prothrombin or clotting time. The Quick method, consisting of the use of sodium oxalate solution, calcium chloride solution, and thromboplastin, has recently been modified by Smith, Ziffren, Owen and Hoffman (for human determinations) as follows:

One-tenth cubic centimeter of thromboplastin (a preparation of macerated rabbit brain extracted with physiological saline solution) is placed in a small serologic tube (75 × 10mm outside diameter). Freshly drawn blood is placed into the tube up to the 1cc mark, and the tube is immediately inverted to allow complete mixing of the blood and thromboplastin. The tube is tilted every second or so to observe clotting. A duplicate test, using the blood of a normal subject, is then run. The calculation is based on the following equation:

$$\text{Percentage of normal clotting activity} = \frac{\text{Clotting time of normal control}}{\text{Clotting time of patient's blood}} \times 100$$

Thus, if the patient's blood clots in 48 seconds and the normal subject's in 24 seconds, the clotting activity is 50% of normal. Such a test is a presumptive measure of the amount of prothrombin present in the plasma—the test measures a summation of the amount of prothrombin and its convertibility. For humans, tests giving values of 40% or less indicates bleeding; and values of 40%-70% are definitely in the danger zone.

Dam and his associates have used a modification of another method for determining the clotting time of avian blood—the modified Fisher method. The sooner one can determine the first signs of impending prothrombin deficiency, the less vitamin and bile concentrates are required for control, and the less serious the danger of loss of blood. The dose for humans has been determined as: 2000 to 4000 Dam units (400mg to 800mg of alfalfa concentrate) with 1gm to 2gm bile as a prophylactic; as a curative dose, 5000 to 15,000 Dam units (1gm to 3gm of alfalfa concentrate) with 2gm to 4gm of bile salts, administered by duodenal tube at various intervals. One or two such doses usually raise the prothrombin level adequately. Until very lately, the oral and duodenal tube route was employed, but recently parenteral methods of administration have been used. However, it has been observed that, contrary to what one might ordinarily expect, the latter method is less

effective than that in which material is supplied via the intestine. As far as experiments have gone, there is no reason to expect vitamin K concentrates to have a favorable effect in other hemorrhagic diseases in which the prothrombin content of the plasma is normal. In cases of sprue or extensive intestinal involvement, improvement may be expected because of the large-scale negative effect on vitamin K absorption. It has been found that purpura, hematuria, and hemophilia are not amenable to such therapy. Functional menorrhagia and recurrent hemorrhagic retinitis have been shown to respond, however. At present, the chief indications are hemorrhagic diatheses due to hepatic and biliary disease.

From the latest findings, it appears that vitamin K is not a single substance but a group of substances which very likely have a synergistic influence on one another. Some recent work has shown the importance of vitamin K to the newborn. The tendency to bleed appears to be great at birth, and losses due to intracranial hemorrhage are very numerous. The administration of vitamin K to the infant at birth or to the mother prior to delivery may at some future date become a routine measure.

In the case of chicks, a 100gm bird requires 0.2mg of vitamin K for proper prothrombin levels. For the present, this must serve as a guide to the use of proper ratios in other animals. The possibilities for using the coagulation-vitamin in veterinary medicine appear to be limited at present, but future application cannot be predicted at this time.

It should be mentioned that several simpler naphthoquinones have been found to possess in varying degrees the antihemorrhagic properties of vitamin K. The most potent of those synthesized to date appears to be 2-methyl-1, 4-naphthoquinone, which in several clinical trials on human patients has in some cases given more satisfactory results than vitamin K itself. Further research and trial are necessary, however, before it can be determined whether vitamin K or one of its substitutes is the agent of choice in controlling prothrombin deficiency.

Or What Have You?

The report of the Chief of the Food and Drug Administration for 1939 makes interesting if not exciting reading. *E.g.* a lead compound ointment which led to the prosecution and fine of the manufacturer, contained on the label the following modest claims as to its virtues: "An effective treatment for frozen feet, eczema, mad dog bite, inflammation of the eyes, blood poison, erysipelas, sore lungs and throat, cuts, sores, all kinds of injuries, rusty-nail sores, burns, boils, carbuncles, diphtheria, gunshot wounds, lame or sore back, caked or inflamed breast, sprained ankle and mumps; and effective to take out and heal all inflammation; to prevent blood poison in man or beast; to heal cuts, wounds and sores; to draw out all the pain, soreness and lameness and to heal sore throat."

The Massengill company, whose Elixir Sulfanilamide was responsible for approximately 107 deaths, drew the heaviest fine during the year—$26,100. Just under $25 for each victim. Possibly the medical profession was not entirely satisfied with the outcome, for we now find their advertisement in a veterinary publication, doubtless on the theory that the veterinarians are less discriminating. But are they?

* * * *

When milk is fortified with small amounts of three mineral elements, iron, copper and manganese, and for children with vitamin D, it can be used as the sole article of diet. At the Wisconsin Agricultural Experiment Station many animals, including rats, dogs and pigs, have grown to maturity and maintained health on a diet of fortified milk alone. Indeed, for three months one winter several men students lived on raw milk supplemented with the missing minerals and with orange juice. They drank as much milk as they wanted—usually three and one-half to four and one-half quarts a day. They maintained their weight, in fact some of the boys gained, and they all felt well and satisfied. As one boy put it, "I can even pass a restaurant and smell beefsteak cooking with no desire to turn in."—*Agr. Exp. Sta., Univ. of Wisc.*

Some Interesting Experiences in Brazil

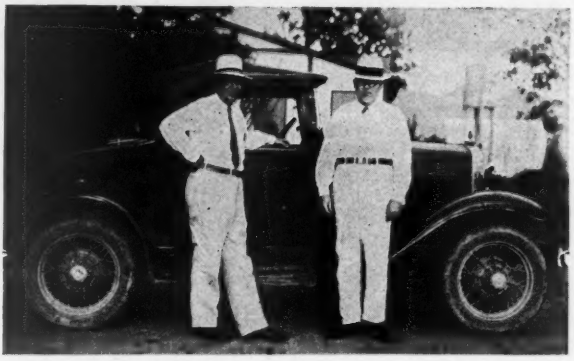
By GUY A. ROBERTS, Ciudad Trujillo, D.R.
Director de Sanidad Pecuaria
Gobierno Dominicano

DURING nine years in Brazil I had many interesting experiences with disease conditions among the animal population, both with those indigenous to the region, and with those appearing for the first time and requiring identification before methods of control could be devised.

One of the most interesting in the latter category was a mysterious disease which appeared among the work oxen of the Wilson packing company shortly after my arrival at Sao Paulo in 1919. The animals affected had been grazing on an area which had previously been used for a shipment of zebus that had passed through Belgium some months before.

The federal veterinary inspector at the plant diagnosed the disease as blackleg. The state veterinarian was positive it was anthrax. To me it appeared more like hemorrhagic septicemia. We were not satisfied with our diagnosis, however, and injected blood from a sick animal into two healthy young steers, one receiving 5cc that had been filtered through a porcelain filter, the other 5cc of unfiltered blood. Both remained healthy for seven days. The eighth day the one injected with unfiltered blood showed a perceptible rise in temperature, the other remaining healthy. By this time, numerous cases were available for study, and a diagnosis of rinderpest (Peste bovina) was made. Dr. W. G. Smillie of the Rockefeller Foundation provided us with equipment and also reproduced the disease.

The characteristic symptoms were: loss of appetite; early loss of flesh; drooping ears; weeping eyes, that left a yellowish stain in the trail of the tears; ulcers covered by a yellowish mucus on the mucous membranes of the lips and at the corners of the mouth; temperature varying from 104°F. to 107°F. After a few days an intense, bloody, fetid diarrhea set in, and death occurred invariably in six to eight days. On autopsy, the most characteristic internal lesion consisted of ulcers in the mucous membranes of the fourth stomach.



The author (left) and A. T. Kinsley. This photograph was taken while they were investigating animal diseases in Puerto Rico in 1932.

The writer was appointed a member of a commission of five to eradicate the disease. The Brazilian veterinarians did a most efficient job, in the face of opposition from the peasantry and even the occasional use of firearms against the work of eradication.

Fortunately, the disease did not break out in any of the other numerous centers to which animals from the Belgian shipment had been sent, but it spread rapidly from the single focus at the Wilson plant, and before repressive measures were applied, an area of some 1,250 square miles was involved. Rigid quarantine measures were established. About six thousand cattle died, and about eight thousand contacts were killed during the six-weeks campaign which finally brought the disease under control.

The hardy zebu is well adapted to the interior of Brazil.

■ Another interesting disease which appeared in southern Brazil during my sojourn there later involved other countries, as far north as the English island of Trinidad, and as far south as the Argentine, where it was known as mal de caderas de los bovinas.

The writer took no part in the solution of this problem, which remained in dispute for several years; in the end, the disease was definitely determined to be a distinct type of rabies transmitted by the bite of hematophagous (bloodsucking) bats. Two species of bats were incriminated. Other than in cattle, the disease rarely appears naturally, but it is inoculable into dogs, horses, and guinea pigs, and presumably into other animals. The type occurring in Trinidad infects both cattle and men, and is inoculable into other animals.

■ Among the diseases indigenous to the region, mal de caderas in equidae, produced by *Trypanosoma equinum,* was common in many sections of western Brazil and Paraguay. Anaplasmosis and piroplasmosis came in for their share of attention among all imported cattle, except zebus, which have a high degree of resistance to these protozoa. Foot-and-mouth disease existed very extensively, recurring in a given locality at intervals varying from one to several years. This disease seriously affected milk production among dairy cattle. Many animals were also rendered unable to walk by a growth between the toes (friera). Treated early with a copper sulphate solution or cauterized with a hot iron, they usually made a rapid recovery.

Infectious bulbar paralysis, pseudorabies or Aujeszky's disease, was often observed in cattle. We were unable to give the affected animals any relief. The disease has been produced experimentally in Brazil in dogs, rabbits, guinea pigs, rats, and swine; but horses, mules, donkeys, and fowl appear refractory.

Pneumoenteritis (bovera) occurred in calves and pigs. The form existing in pigs was designated as "batedera" because of the characteristic thumping of the flanks. Most veterinarians regarded it as hog cholera, but the writer did not agree. A homologous serum analogous to the Dorset-Niles anti-hog cholera serum was produced, but this seemed to give only slight protection. It has since been determined that the disease is not cholera, but a complication of infections.

■ For some years the Brazilians had been developing a pure breed of beef cattle, which they called "Caracu," cara meaning face, and cu, the other extremity. Other than this breed, which was limited at that time, Holsteins held first place near the city for the fluid milk supply. The great majority of cattle in Brazil, however, were zebus. Being hardier than other breeds,

they were admirably adapted to the interior of the country, which was infested with both external and internal parasites, and serious animal infections common.

The screw-worm fly was likewise annoying in wounds and in the umbilicus of newborn animals. Even a drop of blood on an animal, such as might be caused by the bite of an insect, attracts the screw-worm fly.

In the interior of the country, often a hundred miles or more from any road except an ox-cart trail, the common treatment for an infested wound was to pick up a handful of dry, pulverized manure from the cattle corral and dust it in the wound. The result was usually satisfactory.

Canine piroplasmosis was common, particularly among hunting dogs.

■ An interesting parasite observed was the Berne fly, a type of warble fly, *Dermatobia cyanoventris*. It affected cattle chiefly, but not infrequently dogs, rabbits, and even man (especially in the nostrils) were invaded. The adult fly of this species seeks the shade of tall bushes. It rarely, if ever, approaches the host, but lays its eggs on other insects which attack animals, or on the leaves of plants which are ingested by the host animal. The larvae and tumors appeared more often on the sides and legs of the host than on the back. Clearing the bushes and tall weeds from the pastures proved an effective preventive.

The veterinary school near Rio de Janeiro (above) and the animal industry experiment station at Rio de Janeiro (right).

The sand flea, *Pulex penetrans*, picked up in dusty places, was often an unwelcome guest under the toenails of men and in the feet of hogs.

On many horse- and mule-breeding farms (fazendas), it was customary to allow a modified stallion to run with the mares in order to detect those in heat. Using a worthless stallion, a slit was cut in the sheath and the end of the penis pulled through the slit, so that when the penis was erected, it pointed downward. The animal could not copulate but he obtained the desired information.

Work Oxen

Filterable Viruses
With Special Reference to Immunity*

By EDWARD HINDLE, Glasgow, Scotland.
Regius Professor of Zoölogy,
University of Glasgow

AMONG the many remarkable discoveries of recent times those relating to the part played by filterable viruses in the causation of disease must occupy a very prominent place in the history of veterinary science. It is hardly necessary to mention that foot-and-mouth disease was the first human or animal disease shown to be due to an infective agent beyond the limit of visibility, and this discovery was made only at the end of the last century. Therefore our knowledge of filterable viruses has been acquired almost entirely within the last 40 years and indeed, most of it, within the last 20 years.

The methods of investigation used for studying the nature of filterable viruses differ somewhat from those with which most biologists are familiar. Since most viruses are beyond the limit of visibility the methods employed approximate those used by physicists, for just as one is dependent on the inoculation of experimental animals for demonstrating the existence of a filterable virus, so one has to employ various devices to prove the existence of electrons. One makes "coincidence" observations on both viruses and electrons, and creates an hypothesis or theory which will fit in with the experimental observations. The most convenient and generally accepted hypothesis regarding the general nature of viruses is that they are independent living micro-örganisms. It is true that up to the present all pathogenic viruses are capable of multiplication only in the presence of living cells, and they have not yet been grown in artificial culture media like many bacteria and protozoa. However, it must be borne in mind that many other undoubtedly living organisms have also not yet been cultivated in artificial media; for example, the leprosy bacillus, and the great majority of the higher parasitic animals, cestodes, nematodes, etc.

The results of ultra-filtration analysis, ultra-microscopy and high speed centrifugalization have shown that viruses are particulate and that each has a specific size. Moreover, the sizes of viruses range from just below the limit of ordinary microscopic visibility down to only two or three times the diameter of serum proteins, etc., as shown by the following table:

Virus	*Diameter of particle in millimicrons*
Psittacosis	300
Vaccinia	175
Bovine pleuro-pneumonia	150
Herpes	130
Infectious ectromelia	130
Influenza	100
Newcastle Disease	100
Laryngotracheitis	82
Fowl Plague	72
Rift Valley Fever	34
Equine Encephalomyelitis	25
Yellow Fever	24
Louping-ill	17
Foot-and-Mouth Disease	10
Poliomyelitis	10
(Oxyhemoglobin)	3-5

It should be emphasized that the use of graded collodion filters, such as "gradocol" membranes, is essential for determining the size of particles and even to determine whether or not an infective agent is filterable. Porcelain filters, still in common use, present such large surfaces that unless the filterable virus is present in considerable quantity it may be completely absorbed by the substance of the filter. In consequence negative results are not infrequently obtained in attempts to pass undoubted viruses through porcelain filters. The use of the ultra-centrifuge is, of course, confined to those few laboratories in which this

*Presented at the 56th Annual Congress of the National Veterinary Medical Association of Great Britain and Ireland.

somewhat elaborate apparatus has been installed. Ultra-microscopy also demands extremely specialized apparatus and technical skill, and the use of ultra-violet rays in the photography of virus particles requires a microscope with the lenses made of quartz instead of glass. Recently an entirely new method of ultra-photography has been devised, involving the use of beams of electrons, and by this means objects may be photographed far below the limit of even ultra-violet ray photography. (See Krause, 1937, and V. Borries and Ruska, 1938.) In this "electronic" microscope, beams of electrons are straightened out in a magnetic coil, passed through the specimen to be studied and focused in another coil. Since electrons affect photographic emulsions, it is possible to obtain photographs of the magnified objects. The resolving power is said to be 25 times that of the ordinary microscope (0.2μ); therefore, by this new method it should be possible to obtain photographs of virus particles even so minute as those of foot-and-mouth disease.

The X-ray investigation of virus preparations has also given valuable results and indicates the possibility of developing a system of classification on the basis of their X-ray patterns. (Bernal and Fankuchen, 1937.)

Another characteristic feature of viruses is their power under appropriate conditions, of indefinite multiplication, also a characteristic of living matter.

Finally we come to an extremely important characteristic of living organisms, *viz.*, their antigenic individuality and power of variation.

The maintenance of antigenic individuality irrespective of the tissue or animal species in which the virus is grown is of great significance. If a human strain of herpes is adapted to the guinea-pig and an anti-herpes serum prepared, this serum will neutralize specifically not only guinea-pig strains of herpes, but also human and rabbit strains. The same results are obtained by the use of complement fixation.

Similar results have been demonstrated in the case of anti-vaccinia serum, produced in guinea-pigs, which will react specifically with vaccinia virus of guinea-pig, rabbit or human origin. If filterable viruses are unorganized, non-living agents, their multiplication in living tissues must result from changes in the affected cells producing more of the agent which caused the initial change. We should expect an agent of this sort to bear the antigenic imprint of the animal species from which it arose. This is not the case, and to explain this absence we should have to assume that the virus was something in the nature of a haptene which, uniting with the cell degeneration products, gives them a new antigenic value, a virus specificity. A much more reasonable explanation of these facts is the view that viruses are independent, living beings.

To prove the existence of variations in the virulence of any parasite is difficult, for one has to deal with two variables, the parasite itself and the host. The variation of the host may be expressed in many different ways: increased difficulty experienced by the parasite in gaining access to the host; changes in the response of any protective mechanism, such as immune body formation; possible influence of concurrent infections, etc., etc. All these factors and many others have to be taken into account in assessing the significance of any variations in the severity of symptoms or in mortality; this aspect of the problem has to be approached statistically. But there is one type of variation that many viruses have been shown to undergo, *viz.*, the attacking of different types of cells from those normally invaded, which can definitely be attributed to changes in the parasite itself. One of the most striking examples of a change in habit of this nature is shown by yellow fever virus. Normally this virus attacks mainly the liver, producing certain characteristic changes, but if the virus is brought into intimate contact with nerve cells by intracerebral inoculation into mice, it may change its "feeding habits," and acquire the property of attacking nerve cells instead of hepatic cells. Such a virus, with affinities for the nervous system, is known as a neurotropic virus, and many viruses have been shown to be capable of acquir-

ing this property. I shall only refer to them briefly in connection with the light they throw on the general problem of the nature of viruses. Neurotropic yellow fever virus has been maintained in serial passages from brain to brain for hundreds of generations without reverting to the type which attacks the liver, but the reverse process is possible. When neurotropic virus is inoculated into the liver of a rhesus monkey it changes its predilection for nervous tissue and may be found in the liver, spleen and blood, and after a few passages reverts to the ordinary form attacking the liver and only exceptionally the nervous system. (See Findlay, 1937.)

The alteration in yellow fever virus from the form attacking the liver to the neurotropic variety, seems to be more of the nature of a fluctuation than a mutation, for intermediate stages occur, and the fact that reversal of the process can be effected by altering the environmental conditions also supports this view.

Sometimes variant strains of viruses arise spontaneously; the disappearance from this country of smallpox and its replacement by the more benign alastrim may be due to such a phenomenon. Generally, however, variants have been produced artificially by repeated passage of the virus through the tissues of an animal only distantly related to the host. The best known example is, of course, vaccinia virus, formed from the virus of smallpox by passage through the calf and rabbit.

The relationship of these modified viruses to their parent strains is of considerable interest, but their new characters seem to be definitely a response to changed environmental conditions, and in this respect they resemble the larger animals and plants.

I would emphasize that the change from ordinary yellow fever virus to the neurotropic form, is a gradual one; there is evidence of a series of intermediate forms. Moreover, all forms produce cross-immunity, *i.e.*, recovery from any one of them renders the animal immune against the others.

Turning now to variations in pathogenicity, we have to face the difficulty of variations in the host, but it is hardly necessary with the present audience to mention the well-known fact that different epidemics of the same disease may be associated with a different train of symptoms.

I have stressed this aspect of filterable viruses since in the case of plant viruses the infective agent of tobacco mosaic has been obtained in the form of a crystalline protein by Stanley (1935) and there has been a tendency to construe this fact as an indication that this virus is not living. However, Bernal and Fankuchen (1937) have shown that these needle crystals are not really crystalline in the ordinary sense of the word, since they have only a 2-dimensional regularity instead of a 3-dimensional one, and are better described as fibres or para-crystals. Moreover, at least 20 different varieties of this infection are known, yet all those tested have the same size of particles and give similar or identical serological reactions.

Immunity

The immunological aspect has dominated most of the work on virus diseases, and it is obviously of outstanding practical importance. In the majority of virus diseases, recovery from infection is followed by a long-standing immunity sufficient to afford protection against a second attack, and in the case of yellow fever, for example, immune bodies can be demonstrated in the serum even 60 years after the original infection. However, virus infections show considerable variation in this respect and such diseases as herpes simplex, foot-and-mouth disease, and the common cold, have only transient immunity responses. In addition, there are cases of balanced infections where the virus lives in the host without producing any serious damage. As examples of such infections I may mention herpes in man, salivary gland virus in guinea-pigs, Virus III in rabbits, and probably the agent producing the intranuclear bodies, which I have found in the kidneys of sewer rats from various parts of the world (Hindle, 1932). I think it likely that

further investigation will show the existence of many such examples for it is obviously a disadvantage, from a survival standpoint, for a parasite to kill its host.

Even in the case of highly pathogenic infections, one may expect in course of time a gradual diminution in virulence, since strains of lower virulence would have a better chance of surviving than more deadly ones. Subclinical infections must play a very important rôle in the epidemiology of a number of virus diseases, for even in the case of yellow fever, some of the laboratory infections have been so mild as to escape notice and only subsequent blood examination proved their nature. In addition a large percentage of African natives are found to be immune to yellow fever even in districts from which no clinical cases have been recorded.

It is hardly necessary to mention the difficulties met with in the study of such animal diseases as canine distemper and feline distemper, owing to the widespread occurrence of subclinical infections followed by immunity.

Passive Immunity.—The injection of immune or hyper-immune serum has a very restricted application, since the immunity produced is of short duration, not exceeding two or three weeks. However, in the case of measles it has given good results both in preventing the onset of the disease and in decreasing the severity of the attack; and it is said to be of value as a prophylactic in poliomyelitis if given before the appearance of any clinical symptoms.

Active Immunity.—It was formerly believed that infection was essential for the establishment of active immunity against a virus, but it has been proved beyond doubt that in many cases it is possible to immunize by the injection of so-called "killed" virus (*e.g.*, herpes, canine distemper, psittacosis, influenza, equine encephalomyelitis, yellow fever, rabies, etc.). Nevertheless in many instances viruses, whatever method of "killing" is employed, are extremely poor immunizing agents.

Nearly all effective methods of vaccination utilize live virus, and depend on the establishment of a subclinical infection for the production of immunity. This subclinical infection may be produced by the use of one or more of the following methods:

(1) The inoculation of active virus into some tissue where the pathological changes are of little importance. For example, in the immunization of human beings and monkeys against psittacosis by subcutaneous injections of virus; of cattle against pleuropneumonia by the inoculation of virus into the tail; and of fowls against laryngotracheitis by cloacal inoculation.

(2) The inoculation of active virus together with sufficient immune serum to prevent *clinical* infection; but the serum must not be given in excess, otherwise the infection is completely suppressed and no active immunity develops, consequently, strict quantitative control is necessary. This "virus-immune serum" method has been used with success for vaccination against yellow fever, and in various animal infections, such as rinderpest in cattle, swine fever, African horse-sickness, canine distemper, feline distemper, etc.

(3) The inoculation of attenuated virus to produce a mild infection. This method has been used successfully in the prevention of a variety of infections. The best known example is vaccination against small-pox by means of vaccinia. The virus may be attenuated by physical or chemical agents, as in the case of rabies, canine distemper, etc., but the majority of viruses treated by chemicals retain immunizing properties only in so far as any virus is left alive. The "coating" of the virus by mixing suspensions with egg-yolk or vegetable oils constitutes another application of this method and has been used with success for reducing the activity of neurotropic yellow fever virus.

Biologically modified strains of viruses seem at present to offer the most satisfactory means of applying this method. Such strains may be developed by a variety of environmental changes. The commonest method is by growth of the virus in a different tissue from that generally attacked, usually in another species of animal. Neurotropic strains of yellow fever, herpes,

horse sickness and fowl plague have been produced by passage of the respective viruses in the brains of mice, and in all these cases, while the pathogenicity for nervous tissues is greatly increased, the ordinary pathogenicity is reduced. The subcutaneous or intraperitoneal inoculation of such neurotropic strains produces an immunological response seemingly identical with that following the injection of unmodified virus, but usually without the development of any serious clinical symptoms. Such neurotropic variants can also be produced in the brains of the same species affected by the virus, as in the case of vaccinia, myxoma and Rift Valley fever virus in mice; fowl plague in canaries, and yellow fever in monkeys. In such cases it is generally necessary to give an intraperitoneal inoculation of immune serum shortly before the intracerebral injection of virus, in order to prevent the ordinary strain causing death from generalized lesions before the neurotropic strain has had time to develop.

The growth of the virus in the same tissue but in a foreign host sometimes produces variants, as in the classic example of the production of vaccinia from smallpox virus, by growing it in the skin of the calf or rabbit. In this case the pathogenicity for the rabbit is increased, but for primates decreased. The production of fixed rabies virus from the street strain by passage in the brains of rabbits is yet another example.

Variants may also appear when the virus is grown on the chorio-allantoic membrane of the developing fowl embryo. Influenza virus grown in this way has been used for the vaccination of ferrets and mice against this infection.

Similarly, prolonged growth in culture media containing embryonic tissue may lower the pathogenicity of a virus to such a degree that, as in the case of yellow fever and fowl plague, the subcutaneous injection of such strains produces immunization without any other signs of infection. Another method that has been used is the prolonged growth of pantropic yellow fever virus in mouse carcinoma *in vivo*, which greatly reduces its virulence for monkeys.

Finally, reference should be made to the phenomenon of virus interference. It was first noted in the case of certain plant viruses that infection with one strain protected against another strain of the same virus. A similar phenomenon in the case of yellow fever has been described by Hoskins (1936), who found that although rhesus monkeys inoculated with the pantropic virus alone succumbed, they survived when the inoculation of the pantropic virus was followed within a few hours by an injection of neurotropic virus. Findlay and MacCallum (1937) confirmed and extended this observation, but showed that the reverse was not the case, since pantropic yellow fever virus did not protect against the neurotropic strain. On the other hand, a pantropic strain of Rift Valley fever virus also protected monkeys against infection with pantropic yellow fever virus. The protection does not seem to be connected with the presence of immune bodies and it is suggested that the entry of the cells by the milder virus may protect them against invasion by the more virulent one. Since unrelated viruses failed to show this interference effect it is suggested that in the above-mentioned cases, both viruses in some way combine with or use up the same chemical groupings in the cell, and therefore when one virus has occupied the cell there are no free groupings available for the other virus. However, the problem of interaction between two or more viruses requires much further investigation.

In the short time at my disposal I have only been able to refer to a few of the many recent discoveries on this subject, a fuller account of which, together with references, may be found in recent summaries by Burnet, Keogh and Lush (1937), Findlay (1937), and Bedson (1937). The results already obtained in the comparatively short time that viruses have been investigated, would certainly seem to suggest that in the near future methods of control will be found for all virus diseases affecting man and animals.

Failure of Dietary Magnesium Imbalance To Produce Urinary Calculi in Wethers[1]

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EXTENSIVE experimentation has shown that a vitamin A deficiency[2, 3, 4, 5, 6] or a mineral imbalance uncomplicated by a vitamin A deficiency,[3, 7, 8, 9, 10, 11] particularly one of magnesium,[7, 9, 11] may produce urinary calculi in laboratory animals. There are no reports of similar experiments with male cattle or sheep, the sex and species usually affected. Eveleth and Miller,[12] however, have observed a high level of magnesium in the blood serum of lambs in a flock in which about 2% had died from this cause. Although no analyses were made of calculi or feeds, they believed the high blood magnesium was the result of a high dietary magnesium, which in turn produced the stones. Support for this belief is found in the commonly unappreciated fact that some feeds are relatively high in magnesium as compared to calcium and phosphorus. Then, too, magnesium has been found as one of the components of calculi recovered from live stock.[13, 14]

The authors, prior to the above mentioned report of Eveleth and Miller observed an "outbreak" of cystic calculi in a flock of feeder sheep and subsequently investigated the effect of a ration high in magnesium on its production. The results of this investigation, reported below, emphasize the complex nature of the problem.

Experimental

There were 2600 wethers in the flock observed, and approximately 10% were affected with calculi during the feeding period. Only a small number were affected until the middle of January. Beginning at this time, an average of approximately 10 each day showed the symptoms of the disease. This continued for about one week, after which the number gradually decreased, until, after a period of four weeks, only one or two a day were affected. Of interest is the fact that similar losses had been sustained during each of the three preceding winter feeding periods. It was at this time that an opportunity presented itself for an experimental study of the problem.

A preliminary study of the feeding conditions seemed to rule out a vitamin A deficiency, for the entire flock not only had been fed alfalfa hay for several weeks in the fall, but had received yellow corn for two months prior to the time when the largest number were affected. The remainder of the ration during this period consisted chiefly of sweet clover seed screenings and sweet corn silage, a small amount of yellow corn, linseed meal and salt also being fed. After the linseed meal and corn were discontinued, the screenings, silage and salt made up the entire ration. The water supply was from a spring to which the lambs had access at all times. In order

[1] Paper No. 1796, of the Scientific Journal Series of the Minnesota Agricultural Experiment Station.

[2] Higgins, C. C., 1935. Experimental and clinical observations on urinary calculi. *New England Jour. Med.*, 213, p. 1007.

[3] McCarrison, R., 1931. Further researches on stone. *Indian Jour. Med. Res.* 18, p. 903.

[4] Osborne, T. B., and L. B. Mendel, 1917. The incidence of phosphatic urinary calculi in rats fed on experimental rations. *Jour. A. M. A.* 69, p. 32.

[5] Steiner, Morris, Bernard Zuger and Benjamin Kramer, 1939. Production of renal calculi in guinea pigs by feeding them a diet deficient in vitamin A. *Arch. Path.* 27, p. 104.

[6] van Leersum, E. C., 1928. Vitamin A deficiency and urolithiasis. *Jour. Biol. Chem.* 76, p. 137.

[7] Cox, W. M. Jr., and M. Imboden, 1936. The role of calcium and phosphorus in determining reproductive success. *Jour. Nutrition* 11, p. 47.

[8] Cunningham, I. J. and Marion, 1938. Dietary magnesium and urinary calculi. *New Zealand Jour. Sci. and Tech.* 19, p. 529.

[9] Haag, J. R. and Leroy S. Palmer, 1928. The effect of variations in the proportions of calcium, magnesium and phosphorus contained in the diet. *Jour. Biol. Chem.* 76, p. 367.

[10] Ranganathan, S., 1931. Researches on "stone." Studies in calcium and phosphorus metabolism. *Indian Jour. Med. Res.* 19, p. 1.

[11] Watchorn, E., 1932. The effects of excessive intake of magnesium by the rat, especially concerning the factors relative to the production of renal calculi. *Jour. Hyg.* 32, p. 156.

[12] Eveleth, D. F., and T. W. Miller, 1939. High serum magnesium associated with urinary calculi in sheep. *Vet. Med.* 34, p. 106.

[13] Blount, W. P., 1931. Urinary calculi. *Vet. Jour.* 87, p. 561.

[14] Scott, H. P., 1923. Urinary calculi in sheep. *J. A. V. M. A.* 64, p. 221.

to secure a clue regarding a possible mineral imbalance as a cause of the calculi, samples of the screenings and silage were analyzed for calcium, magnesium and phosphorus according to the procedures described by Morris, Nelson and Palmer.[15] The same analyses were also made of a sample of beet tops obtained from the farm. This was done because it had been the experience of the farmer that cases of calculi occurred within a month to six weeks after the lambs had been on a ration of beet tops. In fact, in order to make use of the beet tops, it had been his usual practice, adhered to in the fall of 1937, to feed them until the first appearance of symptoms of calculi.

The data for the calcium, magnesium and phosphorus content of these feeds, in Table I, show that the screenings and beet tops contained more magnesium than phosphorus and were relatively high in magnesium as compared to calcium. The silage, while containing less magnesium than the other two feeds, nevertheless was higher in this element than in calcium or phosphorus. From a determination of the oxalic acid content of the beet tops it was found that only approximately 40% of the calcium could be considered as available. From these analyses, it appeared possible that the calculi formed on the beet tops ration as well as on the screenings-silage ration were the result of high magnesium. This belief was given support from an analysis of stones obtained from one of the wethers, showing that they consisted principally of NH_4MgPO_4.

TABLE I. The Calcium, Magnesium and Phosphorus Content of the Feeds Used During the Incidence of Calculi (Dry Matter Basis)

Feed	Calcium Per Cent	Magnesium Per Cent	Phosphorus Per Cent
Sweet clover screenings	1.55	0.61	0.42
Sweet corn silage	0.41	0.49	0.33
Beet tops	1.72	1.06	0.24

For the purpose of investigating the effect of a high magnesium ration on the formation of calculi, 20 wethers were picked at random from the several hundred remaining on feed at this farm, and brought to the University Farm, February 18, 1938. Until they were started on their experimental rations on March 16, they were fed

Figure 1. Wether No. 15 showing pronounced symptoms of calculi

sweet clover seed screenings, obtained from the farm from which they were purchased, and field corn silage. This silage contained approximately the same percentages of calcium, magnesium and phosphorus as the sweet corn silage. These feeds were given in the same amounts as the lambs had been accustomed to receiving.

The calcium, magnesium and inorganic phosphorus of the blood plasma of each animal, Table II, were determined on composite blood samples taken before the lambs were started on their experimental rations and twice more during the experiment. The composites represented either two or three consecutive days' bleedings. The calcium was determined by the Clark-Collip[16] modification of the Kramer-Tisdall procedure; the phosphorus by the Fiske-Subbarrow[17] procedure, and the magnesium by the Briggs[18] modification of the Denis procedure.

Urine specimens were also obtained from all the lambs before the start of the experiment and four times during the dura-

[15] Morris, H. P., J. W. Nelson and L. S. Palmer, 1931. Quantitative determination of calcium, magnesium and phosphorus in feedstuffs and cattle excreta. *Ind. Eng. Chem.* (Analytical Edition) 3, p. 164.

[16] Clark, E. P., and J. B. Collip, 1925. A study of the Tisdall method for the determination of blood serum calcium with a suggested modification. *Jour. Biol. Chem.* 63, p. 461.

[17] Fiske, C. H., and Y. Subbarrow, 1925. The colorimetric determination of phosphorus. *Jour. Biol. Chem.* 66, p. 375.

[18] Briggs, A. P., 1922. Colorimetric method for determination of small amounts of magnesium. *Jour. Biol. Chem.* 52, p. 349.

tion of the experiment. Table III shows the pH determinations, made by the glass electrode method, and observations as to crystal formation on these specimens.

At the time the wethers were brought to plasma, it was considered that as high a level as met with in feeding practice should be used. Accordingly, a beet tops ration was used as the basis for the formulation of the rations. Lot I, therefore, received suffi-

TABLE II. CALCIUM, MAGNESIUM AND INORGANIC PHOSPHORUS IN 100CC OF BLOOD PLASMA OF LAMBS

		March 7, 8, 9 (Preliminary)			May 4-5 (Experimental)			August 10-11 (Experimental)		
Lot No.	Lamb No.	Calcium mg.	Magnesium mg.	Inorganic Phosphorus mg.	Calcium mg.	Magnesium mg.	Inorganic Phosphorus mg.	Calcium mg.	Magnesium mg.	Inorganic Phosphorus mg.
I ($MgCO_3$)	1	11.49	2.54	6.71	10.37	2.74	6.62	9.49	7.72	6.10
	2	10.40	3.91	7.02	9.23	6.05	6.85	8.91	9.36	6.73
	3	10.50	2.61	6.31	9.58	3.42	6.85	9.13	8.88	5.76
	4	10.81	2.66	8.26	9.63	4.31	7.30	7.89	9.50	6.12
	5	10.97	2.43	7.69	9.55	3.84	8.03	8.87	7.63	5.68
	6	12.11	2.56	7.19	10.92	3.62	5.41	9.96	6.03	5.01
	7	11.18	2.51	7.09	9.48	3.10	6.60	9.03	8.62	5.68
	8	10.92	2.47	5.92	10.42	3.62	5.83	10.43	7.84	5.78
	9	11.54	2.80	8.93	9.78	3.93	7.81	8.40	8.51	6.86
	Mean	11.10	2.72	7.23	9.88	3.85	6.81	8.97	8.96	6.15
II ($MgCO_3$ + $CaCO_3$)	11	10.19	2.49	9.22	9.33	2.99	9.43	9.13	9.01	4.33
	12	10.55	2.51	6.54	9.88	3.41	7.84	10.53	6.45	4.33
	13	11.12	2.79	7.55	10.52	3.07	6.54	10.38	6.51	3.98
	14	10.97	2.48	7.41	10.33	3.17	8.00	10.17	7.72	4.27
	15	11.28	2.31	6.94	9.53	2.85	7.87	9.96	6.69	3.90
	16	10.50	2.58	7.04	9.23	3.25	7.69	9.03	8.37	4.57
	17	11.02	2.73	7.09	10.23	3.28	7.84	9.91	7.11	4.58
	18	10.76	2.38	5.99	9.38	2.80	9.71	9.29	6.62	3.80
	19	11.85	2.66	6.21	10.18	3.48	7.04	9.65	6.62	4.30
	20	11.02	2.30	8.15	9.73	3.62	9.26	9.86	8.52	4.95
	Mean	10.93	2.52	7.22	9.83	3.19	8.12	9.79	7.36	4.30

the university, none showed symptoms of calculi. Two days later, however, one wether, No. 15 (Fig. 1), showed the usual symptoms of this condition. These symptoms have been adequately described by Pontius, Carr, and Doyle.[19] In an attempt to relieve the animal, the process urethra was removed. No calculi were found. During a period of approximately four weeks, this wether was observed to have a total of five attacks. After recovery from the fifth attack, it was not further affected. As can be seen in Table II, this lamb, like the others, had normal values for blood plasma magnesium before being started on the experimental rations.

The 20 wethers were divided into two lots of 10 each. Shortly after the experiment was started, one lamb in Lot I died as the result of an injury. Data for this lamb are not included. Both lots were fed the screenings and silage. Since it was desired to study the effect of a high magnesium ration on the production of calculi, and since the lambs had been receiving a relatively high magnesium ration and yet showed a normal level of magnesium in the blood

cient $MgCO_3$ to provide a daily intake of magnesium equal to that on a beet tops ration. The total magnesium intake for Lot I was approximately three times that of the screenings-silage ration. Lot II received the same amount of $MgCO_3$ and in addition a quantity of limestone so as to give a calcium-magnesium ratio of 3:4. Haag and Palmer[9] found that this ratio was not favorable to calculi formation in the rat. On the moisture-free basis, the ration of Lot I contained approximately 1.0% calcium, 1.5% magnesium and 0.3% phosphorus; the calcium content of the ration of Lot II was approximately 5.1% as the result of the added limestone. The rations were fed twice daily. Water and salt were before the lambs at all times. The lambs were fed the above rations for a period of 154 days, from March 16 to August 17.

Experimental Results

The data in Table II show that the only marked change in the elements of the blood plasma for which analyses were made was in the level of magnesium, which increased approximately threeefold. This marked increase was not an immediate effect, for

only a moderate rise in plasma magnesium was evident at the seven weeks' stage of the experiment. There is evidence that the high calcium ration received by Lot II, on the average suppressed slightly the increase in plasma magnesium, and definitely brought about a decrease in plasma phosphate as the experiment progressed.

two lambs were calculi found. The bladder of lamb No. 7 contained one stone and the kidneys of lamb No. 19 contained three. They consisted of NH_4MgPO_4. It seems probable that they may have been present in the lambs at the time they were placed on the experiment.

This failure to produce calculi on a diet

TABLE III. OBSERVATIONS ON LAMB URINE SPECIMENS

Lot	Lamb	Feb. 24		April 5		May 3		June 9		July 26	
No.	No.	pH	Crystals	pH	Crystals	pH	Crystals	pH	Crystals	pH	Crystals
	1	8.18		8.22		7.88	Crystals	8.15		7.99	Crystals
	2	8.31		8.24		8.23	Crystals	8.22	Crystals	8.00	
	3	7.86		7.76		8.02	Crystals	8.12	Crystals	7.90	
I	4	7.93		8.16		8.11		8.31		7.39	
	5	7.78	Crystals	8.24		8.12		8.07		8.01	
	6	8.11		8.20		8.21	Crystals	8.40	Crystals	7.91	Crystals
	7	7.94		8.31		8.09	Crystals	8.10	Crystals	7.80	
	8	7.41		8.32		8.04	Crystals	8.19		8.75	Crystals
	9	7.74		8.22		8.15		8.21		8.02	Crystals
	Mean	7.92		8.18		8.09		8.20		7.97	
	11	7.40		8.22		8.00		8.19		8.04	
	12	7.81		8.14	Sediment	8.32		8.32		8.30	
	13	7.74		8.31		7.99		8.02	Crystals	7.84	Crystals
	14	7.60		8.33	Sediment	8.11		8.16		8.01	Crystals
II	15	8.35		8.32		8.09		8.06		8.20	
	16	8.13	Crystals	8.35	Sediment	8.18		8.22		8.21	
	17	8.38		8.29		8.19		8.18		8.08	
	18	7.73		8.22		8.50	Crystals	8.12		8.23	
	19	8.21		8.18		8.12	Crystals	8.00		8.11	Crystals
	20	8.25		8.16		7.97	Crystals	8.10		8.11	Crystals
	Mean	7.96		8.25		8.15		8.14		8.11	

No symptoms of urinary calculi occurred. However, after about seven weeks the urine specimens of some of the animals, as shown in Table III, contained deposits of inorganic crystals. The blood plasma magnesium was not exceptionally high at this time, but even when the marked elevation of plasma magnesium had occurred, the incidence of urinary crystallization was no greater. The urine specimens were alkaline in all the tests made, and there was no significant difference between the groups in the mean pH of urine specimens in the several tests. There is indication that the urinary pH was, on the average, slightly higher in both groups as a result of the experimental rations. Thus the experiment lends no support to the belief that a urine of high alkalinity necessarily predisposes to calculus formation. Pontius, Carr and Doyle[19] attach considerable importance to it in spite of the fact that their own results do not sustain their opinion.

After completion of the test, the lambs were slaughtered and a careful examination made of their urinary tracts. In only

sufficiently high in magnesium as to result in a marked increase in blood magnesium duplicates a similar experience of Watchorn[11] with rats. She produced calculi in some of her animals on a high magnesium diet, while in others calculi were not produced in spite of the fact that the experimental diet brought about similar increases in blood magnesium. The only difference in the response of the two groups of rats to the high magnesium diet, other than as concerns calculus production, was diarrhea in the groups in which calculi were not produced. This may have meant, as the author suggested, a fecal rather than a renal elimination of magnesium. This may be of significance in connection with our experiment in that the wethers, while not having what could be correctly termed diarrhea, produced feces which were abnormally soft. Flocks[20] points out that renal calculi in humans is associated with excessive renal excretion of the mineral elements composing the calculi. The variations among different animals in the uri-

[19] Pontius, B. E., R. H. Carr and L. P. Doyle, 1931. Urinary calculi in sheep. *Jour. Agr. Res.* 42, p. 433.

[20] Flocks, R. H., 1939. Calcium and phosphorus excretion in the urine of patients with renal or ureteral calculi. *Jour. A. M. A.* 113, p. 1466.

nary excretion of magnesium and phosphorus on a ration high in magnesium and phosphorus and low in calcium might be worth studying in relation to the incidence of calculi. Our study did not include any mineral balance trials. The failure of any of the experimental animals to develop calculi might have been due to the fact that they were taken from a flock in which very few "susceptible" animals remained.

Summary

In a flock of 2600 wether lambs, approximately 10% were lost from urinary calculi over a period of approximately six weeks. Analyses of stones from a lamb which had the disease and of the ration fed the lambs suggested that the high magnesium content of the ration was the cause of the trouble.

Twenty lambs were purchased from the flock and used in a study of the effect of a ration high in magnesium on the production of calculi. One-half of the lambs received a ration the dry matter of which contained 1.5% of magnesium. The remaining lambs received the same ration plus sufficient limestone to give a calcium content of 5.1%. The rations were fed for 154 days. Urine specimens for pH determinations and observations as to crystal formation and blood samples for calcium, magnesium and inorganic phosphorus analyses were obtained before the start of the experiment and at intervals thereafter.

There was a threefold increase in the blood magnesium of both groups of sheep as the result of feeding the experimental rations. In spite of this increase, which apparently was responsible for crystal formation in the urine of a few of the lambs, calculi were not produced.

Iron Deficiency in Cattle in the Tropics

There is a very puzzling condition affecting cattle here in the West Indies.

Most of the animals affected are reared in a neighboring island about 12 miles away. They are in the majority of cases wild range animals, i. e. they have never been stabled and run on pasture all the time.

While they are on the range they look in good condition. After they have been caught and brought here for working purposes, they are fed on crushed linseed meal and cane molasses mixed with cane tops cut up in the form of chaff, and in the reaping season cane tops in a green state and fed whole—called locally long tops to distinguish it from chaff—in addition to chaff; and when guinea grass is available, from about May to December, they get some in addition to chaff with linseed meal, etc.

Most of these animals are young, about four to eight years, usually about four to six years of age when affected.

Here comes my problem: many of these animals before they begin to do any work, gradually lose condition, getting thinner and thinner until after a variable period, from three months to a year, they are unable to rise and will remain lying down for weeks, eating and chewing the cud until they succumb. Some develop a submaxillary edema.

On post-mortem examination, the blood is small in quantity, thin and watery. The lungs are pale, heart muscle pale, the liver is a sort of buff color and so are the kidneys. The spleen in some cases is normal and in others has a feeling of emptiness. The lymph glands are shrunken and from the cut surface serum exudes.

Home Ranch with 14,000 Sheep, Snake River Valley, Idaho.

Sometimes the lungs contain a large number of Dictyocaulus, and the abomasum *Haemonchus contortus*. Sometimes nodular disease is found, and sometimes scattered along the small intestines black spots ranging in size from that of a pinhead to that of a split pea.

Microscopic examination of the blood and intestinal contents by a trained bacteriologist fails to disclose organisms.

The condition is not confined exclusively to the animals coming from the neighboring island.

I am not sufficiently experienced to state positively if the number of parasites present is responsible for the condition. It seems rather strange that the parasites, if present when the animals are on the range, which is most likely, do not affect them under those conditions, and yet seem to have a deleterious effect when the animals are kept, one would think, under better conditions.

Is there some mineral in the soil which the animal is deprived of under changed conditions? I have read that in young calves hoose is a serious condition and so is a heavy infestation of *Haemonchus contortus*, but in many of these cases, I would hesitate, from the number of worms found on postmortem examination, to state positively that parasites are responsible for the death.

I have tried various tonics, iron, Fowler's solution, copper sulphate, gentian, and a general so-called condition powder of my own formula, composed of iron sulphate, potassium nitrate, sublimated sulphur and powdered nux vomica, ginger and gentian without much improvement.

I shall be very grateful for any advice.—*E. F. J., St. Kitts, British West Indies.*

Comment: This description resembles somewhat a condition rather frequently encountered in St. Croix, Puerto Rico and the Dominican Republic. It likewise has much in common with that described by Shealy in Fla. Bul. 231, 1931.

The cases we have observed have nearly all occurred on rather low-lying land during a dry season. They occur in cattle of all ages but with more frequency in mature animals than in calves. We at first thought it possibly due to parasites, which may produce a similar picture and no doubt play an important role. However, most parasites alone are usually much more detrimental to the young than to the adult.

We next thought the trouble due to lack of phosphorus, but in comparing the bones of animals dying from the disease with those from normal animals we noted no difference.

At last, in noting the lowered hemoglobin content and limited worm infestation, we concluded that the condition was due to iron deficiency. It was further noted that when such cattle were removed to greener and more luxuriant pasture, especially to higher ground, no new cases developed and most of the affected animals improved and recovered.

One not familiar with the difficulty of teaching old dogs new tricks will be surprised to see adult cattle, not accustomed to grain feed, starve before touching ground corn and bran. They will often refuse cut grass, fodder and even salt, if medicated.

One case of interest was a young cow with her first calf. When first observed she was very thin and weak, and had difficulty in getting on her feet when down. She refused to eat grain or medicated grass, and also refused Shealy's prescription of 100 pounds salt, 25 pounds red oxide of iron and one pound copper sulphate as a "salt lick." She was therefore given as a drench twice daily red oxide of iron and copper sulphate in a gruel of linseed meal, corn meal, bone meal and molasses water. This was continued for nearly a month before rains fell and grass became luxuriant. She was then turned to pasture and made a complete recovery, only to come down again the next year, when, without treatment, she died.

To come back to the case in question, we believe the trouble to be due in the main to iron deficiency in the feed. Parasitosis and perhaps avitaminosis of some sort play a part. Prevention should be attempted by changing pastures or source of feed. Treat with red oxide of iron and copper sulphate in the most convenient manner.—*G. A. R.*

Pulmonary Edema in Hogs

By L. E. STARR, Auburn, Alabama
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Alabama Polytechnic Institute

PULMONARY edema in hogs was reported by A. T. Kinsley in VETERINARY MEDICINE, March, 1937, p. 146. According to Kinsley, the disease is characterized by an accumulation of fluid in the intercellular tissue spaces in the lung. He suggested that there might be some relation between pulmonary edema in swine and influenza.

During the past year 17 pigs have been presented to the Pathology department for diagnosis by the Animal Husbandry department of this institution. All showed pulmonary edema as the cause of death. The first pigs examined post mortem were suckling pigs and weighed 15 to 25 pounds. They were maintained in hog lots which had been used by hogs for many years. One entire litter of seven pigs died. The remaining pigs in the litters were placed on a feeding experiment. Each had an individual pen with a concrete floor which was cleaned daily by flushing with a hose. They were fed in individual troughs so that there was no contact from pig to pig except by the feet of the attendants. Over a period of 90 days, seven pigs died of pulmonary edema.

The etiological agent was not determined, although the history indicates that it was a specific infection. Kinsley states that the malady is a toxemia, but if so that it is probably the result of a bacterial or virus infection.

Symptoms

The symptoms were depression, accelerated breathing, and dullness over the thoracic cavity, with anorexia in some cases. The pigs usually appeared to be normal until three or four days before death. The temperature of those observed before death was approximately 103° F. Dyspnea was very marked in the final stages.

The lesions found at necropsy included extreme edema of the entire lung. The trachea and bronchi were completely filled with a foamy mass. The tissue interstices in the mediastinal space and interlobular spaces of the lung were filled with fluid. The lungs had a grayish color, did not collapse, and had a doughy, water-bag consistency. They were full and rounded, and sank in water. Upon incising the lungs the peribronchial tissues as well as the bronchi themselves and apparently the alveoli, were filled with fluid. The fluid was not organized nor was there any evidence of necrosis or abscess formation. All parts of the lungs were extremely edematous, apparently resulting in the drowning of the animal in its own fluids. There were a few ounces of fluid in the thoracic cavity.

Diagnosis

Diagnosis of the condition can be made readily upon post mortem examination, as the edema of the lungs is very striking. Diseases such as hemorrhagic septicemia, influenza, parasitism and atypical catarrhal pneumonia are usually accompanied by a certain amount of edema and ascites. The pulmonary edema described appeared as a mild epizootic with the macroscopic lesions confined entirely to those of uncomplicated edema. It would appear, therefore, that we were dealing with some specific bacterial or virus disease.

Prevention

In the absence of a known etiological agent we can advise only that sanitary conditions be improved as much as possible, with prompt isolation of the sick animals. Well animals should be moved to clean quarters, preferably quarters that have not been used by hogs for some length of time.

Considering the nature of the disease, treatment would probably be unsatisfactory because the edema is well advanced before clinical symptoms are manifested. In the absence of a known etiological agent, bacterins or other biological products are probably not indicated. In the outbreak described no treatment of any kind was used. Other litters of pigs have since been raised in the same pens without a recurrence of the disease. Whatever the nature of the disease, it appears to be self-limiting.

Infectious Necrotic Enteritis

By A. T. KINSLEY, Kansas City, Missouri

FROM the economic point of view, infectious necrotic enteritis is the most important disease of swine in this country. It is also prevalent in some foreign countries. It may occur in recently farrowed pigs or in mature hogs, and swine of all breeds appear to be equally susceptible. It is apparently more fatal in stocker hogs that are treated with anti-hog cholera serum and virus and shipped immediately considerable distance. The estimated mortality in such hogs may be 50% or even higher. The disease is usually more fatal and more prevalent in the spring time than in the fall. Various predisposing influences such as improper feed, insanitary surroundings, and undue exposure diminish the resistance of swine and, no doubt, increase their susceptibility to infectious necrotic enteritis; however, it may occur regardless of the condition or surroundings of the swine.

Cause

The primary cause of the disease is *Salmonella suipestifer*. Various bacteria are usually associated with this organism, may be of some etiological significance in this malady, although the disease has been produced by Murray and others by feeding pure cultures of *S. suipestifer*. This organism is of common occurrence, but it may occur in apparently healthy swine, and is, therefore, probably not strictly pathogenic.

The virulency of *S. suipestifer* is variable and is apparently increased by passage through swine. Since infectious necrotic enteritis can be produced experimentally in healthy hogs by feeding cultures of *S.suipestifer*, and the infective microorganism can be demonstrated in the feces of diseased swine, it is probable that the usual source of infection is contaminated feed and water; feed lots, sleeping quarters, and feed and watering troughs, etc. The length of time that *S.suipestifer* will remain alive in soil, feed, and water has not been determined.

Clinical Symptoms

The symptoms of infectious necrotic enteritis are variable and their intensity frequently is not in proportion to the intensity and extent of the lesions. Swine that are apparently healthy may reveal extensive lesions indicative of this disease when slaughtered, while swine manifesting marked symptoms may show very limited lesions on necropsy. Although infectious necrotic enteritis was formerly considered primarily a chronic or subacute malady, there have been many outbreaks of the acute type reported recently in both this country and Europe.

The acute form (septicemic) is commonly designated paratyphoid. It is characterized by a sudden onset, high temperature (105° to 107°F.), inappetence, arched back, frequent urination, and a blue discoloration of the belly. Usually it affects a large percentage of the herd simultaneously, and the mortality is high. It is this type of the disease that is produced when cultures of *S.suipestifer* are fed experimentally.

In the subacute or chronic type of the malady, properly designated as infectious necrotic enteritis, only a few animals in a herd are affected at a time. They die or recover, and later others in the herd become infected, the disease thus ultimately affecting practically all the swine on a given farm. The chronic form is manifested by depression, altered appetite, temperature up to 105°F., and a tendency for the affected swine to separate themselves from

the other hogs. As the disease progresses, digestive derangements are manifested by irregular appetite and diarrhea. At this stage the affected hogs appear unthrifty, have rough coats, arched backs, and begin to lose weight. The fecal discharges are quite fluid, and of variable color, depending upon the feed. The animal becomes emaciated, thin and weak, and may die of exhaustion. The disease is some times called "drying up disease" by feeders and breeders because of the extreme emaciation manifested by some affected individuals.

Lesions

The acute type of necrotic enteritis, that is, the septicemic form, is characterized by enlargement of the spleen, hemorrhages and tumefaction of the lymph glands, particularly of the digestive viscera, usually some small hemorrhages in the kidney cortex, and nephritis. There may be also hemorrhages or acute initial inflammatory changes in the mucosa of the small and large intestines.

The principal lesions of the chronic type of infectious necrotic enteritis occur in the mucous membrane of the intestine. In some cases the lesions indicate that the infection begins in the lymphoid areas of the intestinal mucosa. Lesions may be found in any portion of the digestive tube from the mouth to the rectum, but they are usually more extensive in the intestine. In the beginning the lymphoid areas become congested and tumefied, and later they undergo necrosis. In other instances the infection appears to invade the mucosa, which becomes congested, tumefied, and undergoes necrosis. When large areas of the mucosa become involved, the accumulated adherent exudate and necrotic mucosa appear as a diphtheritic inflammation. The entire intestinal wall may be thickened due to the accumulated exudate and necrotic debris. The necrotic material may slough, leaving denuded areas of varying sizes.

In the prolonged cases of this disease, the so-called "button ulcers" are prone to occur. This lesion apparently occurs when the infection is localized in the lymphoid tissue (solitary follicles and Peyer's patches) and the inflammatory and necrotic processes extend by continuity, the necrotic tissue remaining intact or accumulating as dirty brown masses with raised margins. Microscopically, the epithelium and lymphoid tissues in the "button ulcers" are found to be in various stages of degeneration and disintegration. The degenerated and dead cells commingle with the coagulated inflammatory exudate and appear upon the surface as an adherent mass.

The mesenteric lymph glands through which the lymph passes from the infected mucosa are usually tumefied and may be congested and hemorrhagic. In long-standing cases the central portion of the related lymph glands may be necrotic. The peritoneum adjacent to the involved intestine is usually affected with a subacute or chronic inflammation which is manifested in the early stages by an outpouring of serous fluids, but as the process continues, there is a tendency to organization and limited fibrous proliferation which is prone to produce adhesions.

Diagnosis

The acute or septicemic form of this disease is characterized by sudden onset, with a large percentage of the herd affected simultaneously, high temperature, inappetence, blue belly, enlarged spleen, and nephritis.

The chronic form is characterized by dullness, depression, irregular appetite, diarrhea, emaciation, weakness, and inflammatory necrotic lesions of the intestinal mucosa.

In making a diagnosis it is always advisable to consider the history of the outbreak in conjunction with the symptoms and post-mortem findings. Differential diagnosis involves a consideration of hemorrhagic septicemia, hog cholera, and swine erysipelas.

Control

Insanitary pens, overcrowding, intestinal parasitism, and improper feeding diminish the resistance of swine and favor the spread of infection. There is a tendency for the larger feeders to keep too many hogs in a lot, a practice that is conducive to the rapid spread of infections. Rigid enforcement of quarantine regulations, including proper disposal of the feces in the early stages of infectious necrotic enteritis, will materially diminish the losses. Shipped-in stocker pigs are prone to develop this malady and should therefore be effectually quarantined for at least three weeks after arrival.

Infectious necrotic enteritis can be controlled and the losses reduced by providing sanitary pens and lots, proper housing and feeding, elimination of ascarids, and the establishment of an effective quarantine. All excreta and carcasses of dead swine should be collected daily and properly disposed of by liming, burying or cremating, for the feces carry *S.suipestifer*, the causative factor of the disease. *S.suipestifer* does not inhabit the intestine of normal swine; its presence therefore indicates a diseased animal or a carrier.

Prevention

Although reported experiments are not all in accord, field tests indicate that at least a temporary immunity is produced by a bacterin made of *S. suipestifer*. The commercial bacterins that have been used in the prevention of this malady have been in most instances a combination of *S.suipestifer* and various other microorganisms that are found in the lesions of necrotic enteritis. It is quite probable that better results will be obtained in the prevention of this disease by the use of a specific suipestifer bacterin. Extensive losses from this disease in stock hogs simultaneously immunized against hog cholera in public markets have been materially diminished by the use of suipestifer bacterin. The administration of suipestifer bacterin to all exposed swine on a farm is advisable, and some practitioners claim to have produced beneficial results by the use of this bacterin as a curative agent.

Treatment

As a rule, swine affected with infective necrotic enteritis do not respond readily to treatment. Affected swine should be provided with easily digestible slop foods and placed in comfortable quarters. Some garbage feeders have observed that those swine that consume quantities of citrus fruits are rarely affected with infectious necrotic enteritis, and others claim that the feeding of citrus fruit refuse to swine in the early stages of the disease is very beneficial. Some veterinarians report the successful use of acetic acid in quantities sufficient to acidulate the slop. The basis for this treatment is the fact that neither the *S.suipestifer* nor the *Actinomyces necrophorus*, a frequent secondary invader in necrotic enteritis, thrive in an acid medium. However, acidifying the intestinal content by the ingestion of acids entails an increased alkalinity. Other practitioners have obtained good results by the use of caustic potash in sufficient quantities to render the slop distinctly alkaline. Here again it is doubtful if the reaction of the intestinal content is materially altered, since the alkaline slop would merely increase the flow of hydrochloric acid in the stomach, by which is would be neutralized.

The use of intestinal antiseptics is indicated; the antiseptic selected should be such as can be administered in the slop. Copper sulphate in one- to two-dram doses daily either in the slop or in capsules is probably of value.

The intravenous injection of 50 to 100cc of freshly prepared proflavine and acriflavine in cases of the acute type of the disease has produced some splendid results and is worthy of trial when the value of the affected swine justifies the expense.

Icterus in a Garbage-Fed Hog Due to a Foreign Body*

By G. C. HOLM, Moscow, Idaho
Animal Pathologist,
Bacteriology Department,
University of Idaho

ONE of Dr. E. T. Baker's clients brought a hog to this laboratory for post-mortem studies. The herd from which this animal was taken had been fed on garbage for about three months, had shown no evidence of infectious or nutritional disorders, and had been making satisfactory gains. The pig in question had suddenly lost its appetite and began losing weight rapidly. It was isolated and observed for a few days before bringing it to the laboratory for autopsy.

The foreign body. A, the larger mass, was wedged into the pyloric part of the stomach; B, the smaller mass, was in the jejunum.

Doctor Baker aptly described its woeful bewilderment by stating that it looked like a neophyte in the midst of rough week. The animal showed a marked icterus on all mucous surfaces. It had a temperature of 103°F. and was extremely weak. During ante-mortem examination the pig made several attempts to vomit.

The striking condition observed post mortem was the presence of free bile in the peritoneal cavity. The intestines appeared to be highly inflamed, with the loops apparently adhering to each other. Closer inspection disclosed that some object was holding the loops of intestines in close apposition to the pyloric part of the stomach. Palpation revealed a hard, cord-like object ranging from the pyloric opening of the stomach posteriorly for about a foot. An enterotome was used to lay the intestine open. At the posterior or free end of the cord a wad of rags (B) was found; this object was in the jejunum. The intestines were laid open along the cord anteriorly, revealing a severe enteritis and four intussusceptions. Near the anterior end of the duodenum the foreign body was larger in diameter and had completely occluded the bile duct as well as the pylorus and proximal portion of the duodenum. The mass (A) was tightly wedged into the pyloric opening. After removing the foreign body other visceral organs were examined. The liver was a deep orange-yellow color. The gall bladder had ruptured and its torn edges indicated that the rupture was ante mortem rather than post mortem. The pressure resulting from complete occlusion of the bile duct was great enough to burst the bladder. The spleen and kidneys showed a marked hyperemia and some discoloration from the free bile.

Measurements of the foreign body showed that the cord connecting the two masses (A) and (B) was ten inches in length. The foreign body had no doubt served time as a dish rag or floor mop. The telescoping of the intestines was due to peristaltic action on the mass (B). This mass was about the right size for good peristalsis. Since the cloth could not move posteriorly, the intestines moved forward over the cord.

Eight feet and three inches of intestine were telescoped over the ten-inch cord. Of the four intussusceptions observed, one was six inches long, another five, and the remaining two slightly less than four.

* Published with the approval of the Director of the Idaho Agricultural Experiment Station as Research Paper No. 186.

Submaxillary Cyst in a Shepherd Dog

Of the 2,700 dogs presented at the veterinary clinic of Kansas State College for treatment during 1939, in only three was a

The patient when presented at the clinic.

diagnosis of submaxillary cyst made. This indicates the comparative rarity of this condition.

A submaxillary cyst is a bladder-like sac, containing a viscid material secreted by its lining membrane. It may be due to the dilation of an obstructed duct, but is more often the result of degeneration or of the obstruction of the outlet of a mucous gland. It is sometimes difficult to distinguish between a cyst and an abscess, as both fluctuate under pressure; the chief differentiating features are the absence of heat and pain in the former, and the comparative slowness with which it forms. A cyst, too, has sharply defined edges. An exploring trocar or needle, used cautiously, is very useful as an additional aid to an exact diagnosis.

February 21, 1940, a shepherd dog with a large fluctuating swelling in the inter-mandibular space was presented at the clinic for treatment. A diagnosis of a submaxillary cyst was made. The area over the cyst was shaved and the skin painted with tincture of iodine. General anesthesia was accomplished by the intravenous injection of 9cc of nembutal (1cc per 5 pounds body weight). An elliptical incision was made through the skin down to the lining of the cyst. The removal of the cyst was attempted by carefully and completely dissecting around its external wall, endeavoring to remove it in its entirety without evacuating the contents. A blunt-pointed director was inserted between the skin and the cyst wall, as this is of the greatest service to the operator in removing the cyst lining intact. Complete dissection was impossible in this case because of the proximity to the large blood vessels. The edges of the skin were then sutured and the cavity packed with gauze saturated with tincture of iodine, which was withdrawn at the end of 24 hours.

Daily treatment consisted of cleansing the external wound. The first four days following the operation the cavity was swabbed with gauze treated with tincture of iodine. By this time healthy granulation tissue had started to form in the bottom of the cavity. The sutures in the skin were then removed and the cavity was allowed to heal as an open wound. It was again treated with tincture of iodine on the 8th and 12th days.

Rapid recovery was made and the animal was discharged on the 15th day. During the period of treatment the patient showed no inappetence, nor was the temperature elevated.

WALTER F. MANINGER, K. S. C. '40.
Manhattan, Kans.

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Rabies in the Army

Single-dose anti-rabic vaccine was given 6620 small animals (6303 dogs, 305 cats, 12 monkeys) owned by military personnel in or near Army stations during 1938.[35] Four cases of rabies occurred in this group; 15 cases were reported among non-vaccinated animals, of which 13 were strays or civilian-owned dogs. One horse and one mule died of rabies at Fort William McKinley, P. I., after being bitten by a rabid stray dog. The chief source of exposure of military personnel is the non-vaccinated stray or civilian-owned dog. The vaccination of small animals owned by military personnel seems to be lowering the incidence of the disease in the non-vaccinated animals in the vicinity of the military stations.

[35] Rabies in the Army—1938. The Army *Vet. Bul.* 33:2, pp. 133-134.

Differentiation of Viruses of Canine Distemper and Fox Encephalitis

By R. G. GREEN, Grafton, Wisconsin

CANINE distemper and fox encephalitis are often met with in epizoötic form on fox farms. Both diseases can be transmitted to dogs. Distemper in foxes is caused by the same virus that produces distemper in dogs. Epizoötic fox encephalitis has been recognized as a common disease of foxes but has not been recognized clinically in dogs. The virus of fox encephalitis has, on several occasions, been isolated from dogs; and upon experimental test, dogs are found to be highly susceptible to the virus of this disease. Thus it seems that the virus of fox encephalitis may not infrequently produce infection in dogs. Following is a brief description of these two diseases and their main points of differentiation.

1. *Canine distemper* is a virus disease. Dogs are most commonly susceptible to it, although the fox, raccoon, mink, and ferret, also, are highly susceptible and suffer epizoötics both in the wild and in captivity. The disease in all the species mentioned is characterized by inclusion bodies, usually cytoplasmic but occasionally also nuclear, in the epithelium of the respiratory and intestinal tracts, in the bile ducts, kidney pelvis, and urinary bladder.

The distemper virus is modified by host-passage; and a modified virus highly virulent for dogs and foxes is much less so for minks and ferrets. Contrariwise, a distemper virus highly pathogenic for ferrets and minks usually produces milder infections in foxes and dogs. By artificial passage through ferrets, the distemper virus can be modified so completely that, while ultrapathogenic for members of the weasel family, it will produce almost symptomless infections in foxes and dogs. The modified virus is called a distemperoid virus.

2. *Epizoötic fox encephalitis* is a virus disease chiefly of the red or silver fox, although it is rather highly virulent for the dog and the wolf. Ferrets and minks are immune. Clinically the experimental disease in dogs is strikingly similar to uncomplicated canine distemper, except that the duration of symptoms rarely exceeds five days. Epizoötic fox encephalitis is characterized by inclusion bodies, always nuclear, in the endothelium of the smaller blood vessels and by similar inclusions in the hepatic cells of the liver cords.

DIFFERENTIATION OF THE VIRUSES OF CANINE DISTEMPER AND FOX ENCEPHALITIS BY INCLUSION BODIES

	Distemper	Encephalitis
Location in the cell	Cytoplasmic and intranuclear, the cytoplasmic more common than the nuclear.	Intranuclear, never cytoplasmic.
Cells in which inclusions occur	Primarily epithelial.	Primarily endothelial.
	Vascular endothelial cells of the brain never contain inclusions.	Vascular endothelial cells of the brain always contain inclusions.
	Epithelial cells of the urinary tract contain inclusions in 90 per cent of cases.	Epithelial cells of the urinary tract never contain inclusions.
	In the adrenal, inclusions are common in cells of the medulla, but are not found in cortical or endothelial cells.	In the adrenal, inclusions are occasionally present in endothelial or cortical cells but have not been found in glandular cells of medulla.
Staining properties	More acidophilic than encephalitis inclusions.	Acidophilic but less so than distemper inclusions.
Internal structures	Vacuoles common in cytoplasmic inclusions. Both cytoplasmic and intranuclear inclusions less granular than the inclusions of encephalitis.	No vacuoles. More granular than distemper inclusions.

Tumors of Extradental Origin in Dogs

After having several cases of neoplasms of the mouth and lips presented for treatment, I realized that little has been published on tumors of this type in dogs, and resolved to make a study of them.

The tumors of extradental origin are: epulis, osteoma, fibroma, giant-cell tumor, sarcoma and carcinoma.

Epulis is an inflammatory tumor originating from the mucous membrane or the periosteum of the gingival pyramid surrounding the tooth. The exact etiology is unknown; however, two factors have stood out in all cases reported in human medicine—trauma and infection. Karsner states that the giant cell epulis appears as a rule in the alveolar processes of the jaw, usually after extraction of teeth. Infection from a carious tooth or following extraction is almost universally observed, and, as with humans, the epulis may be adjacent to an imperfectly-fitting piece of dental work which causes trauma or pressure. These tumors usually develop in adult animals. I observed an epulis in a five-year-old wire haired fox terrier with heavy incrustation of tartar; it appeared to me that infection was the cause of the growth.

The color of the growth resembles that of the adjacent gum, but as it enlarges it assumes a darker or greyish appearance. This is especially true if ulcerations occur and the growth is traumatized by the opposite grinding teeth. The tumor begins to soften as ulceration occurs. When softening takes place an epulis resembles an early malignant growth by its cauliflower appearance. Large epulides may displace sound teeth, and produce ulceration and necrosis of the gingival tissue. When ulceration and deformity are observed, malignant change is unquestionably present.

Epulides have been observed in pregnant women, due to some calcium disturbance; they disappeared at the termination of pregnancy. Clinicians have also seen epulides occur in hypercalcemic conditions independent of pregnancy (osteitis fibrosa cystica).

According to Rywkind there are three stages in epulis: (1) simple granuloma, the inflammatory tissue stage; (2) angiogranuloma, the stage when vascular elements appear; (3) giant-cell epulis, a combination of the first two stages with giant cells.

The rapid growth of these tumors differentiates them from fibromas.

Treatment of uncomplicated epulis consists in excision of the granulating mass and the involved mucous membranes as well as the periosteum. When the epulis is associated with ulceration and deformity of the jaw, complete excision and cauterization should be carried out. In humans this is followed by irradiation, but with animals economy and feasibility must be considered. However, desiccation or cauterization is indispensable.

Extradental Tumors in a Dog.

Fibroma is a rather rare tumor of the lower jaw. It occurs as a central tumor surrounded by a shell of bone. It usually extends toward the oral cavity, so that as the tumor increases in size, there is a protrusion of the inner wall of the ramus. Occasionally these tumors break into the oral cavity, and are then believed to be periosteal or fibrosarcomatous. The cause of this type of neoplasm is unknown. They are painless, slow-growing tumors which may occur in young or old animals. They may be cured by complete extirpation, even when they have penetrated the bony wall. They are not encapsulated, but are definitely separated from the bone and are not attached to the periosteum. Following com-

plete extirpation, electro-desiccation should be employed. Sufficient bone to maintain the architecture of the jaw should be left. Recently a fibrosarcoma on the lower jaw of a four-year-old scotty was successfully treated at my hospital by this method.

Giant-cell tumor is a central growth of the lower jaw, and must not be confused with the malignant epulis or giant-cell sarcoma of the jaw. The giant-cell tumor usually springs from the angle of the jaw, although other sites have been reported. In humans, these have been diagnosed in patients from 20 to 30 years of age. They are seldom malignant, but frequently recur after improper or insufficient surgery. They increase in size at the expense of bone and grow rapidly, more so than the central fibroma. They are usually found on the lower jaw; rarely on the upper. They are painless. The rate of growth is rapid. Treatment is like that of the other types of tumor, in that they must be completely removed; thorough exposure of the tumor cavity, followed by complete extirpation of the mass, with electro-thermic desiccation of the walls, is necessary.

In general, irritation and infection seem to be the most frequent causes of extradental tumors. Surgery followed by electrodesiccation has proved the most thorough and most successful method of treatment. Treatment with iodine, internally and externally, which is the common method in use by practitioners who dislike to resort to biopsy, is almost useless.

New Rochelle, N. Y. J. J. WERNER

Medial Luxation of the Elbow Joint in a Collie

Fractures of the various bones of the forearm readily lend themselves to reduction and alignment. Luxation of the elbow joint, especially medial luxation, is not so easily dealt with, partly because of a common lack of understanding of the anatomical arrangement of the elbow joint of the dog, which is not entirely analogous to that of the horse and other animals.

The articular surface of the humerus resembles an oblique trochlea of which the medial side is much the larger, the medial condyle being very prominent and the groove deep. The articular surface of the radius is equivalent to about the lateral two-thirds of that of the horse. On the luna the articular surface is prolonged downward and forward on the medial side, articulating with the medial condyle of the humerus as well as with the groove extending into the olecranon fossa. The mechanical arrangement is such that luxation is more likely to occur in the lateral direction, especially in the flexed position.

Case Record.—December 25, 1939, an eight-year-old male collie was presented for treatment at the veterinary clinic of Kansas State College. The owner had observed that morning that the dog refused to use the left front leg, carrying it in an unnatural position.

Temperature, pulse and respirations were normal and the animal was in excellent condition. The left front leg was fixed, deviating outward below the elbow joint at an angle of about 30°. Palpation showed the elbow joint to be rigid and enlarged, but there was no crepitation, nor did the animal evidence pain.

A tentative diagnosis of luxation of the elbow joint was confirmed when fluoroscopic and x-ray examination disclosed a medial luxation of the joint (see illustration in preceding column).

The animal was given an anesthetizing injection of nembutal in the radial vein, and placed on the operating table in lateral recumbency with the injured leg uppermost. An unsuccessful attempt was made at reduction with the joint excessively flexed; which seemed the most plausible method after a skeletal study. When it became evident that reduction in the flexed position was impossible, a wide towel was passed around and under the humerus, resting in the axillary space, and an assistant grasped the towel and applied

traction in a dorsal direction to offset the pull supplied by an assistant who grasped the metacarpus and pulled in a distal direction. As the dislocation was of recent origin, it was not necessary to break down any adhesions. The operator moved the elbow joint in a circle, using his thumb and forefingers, and was successful in guiding the articular surfaces of the humerus into place. The joint immediately became freely mobile and reduced to normal size.

The leg was powdered with boric acid, special care being taken to put small cotton puffs dusted with boric acid between the toes, and was bandaged with a generous length of three-inch muslin bandage, extending as high as possible and including all of the lower part of the limb to prevent edema of the dependent portion. The bandaged leg was covered with water glass, making a waterproof, light-weight, rigid cast.

Fixation for 10 days was sufficient. The strained and torn ligaments healed rapidly and in 30 days the only evidence of lameness was a slight limp after running.

J. L. HOURRIGAN, K.S.C., '40.

Manhattan, Kans.

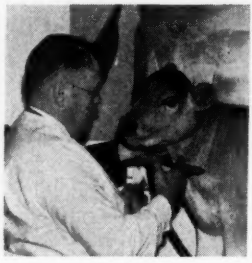
The Lure of the Extra Profit

The Marinan Supply Co., St. Paul, Minn., was found guilty April 12th on two counts charging the shipment interstate of anti-hog cholera serum not prepared in accordance with government regulations. One shipment was made to a veterinarian in West Fargo, the other to a farmer in Sioux Falls, N. D.

In view of the fact that this is the second company caught distributing diluted serum within a few months, it appears the part of wisdom for veterinarians to observe great care in their purchase of serum—in short, to buy only from members of the Associated Serum Producers and take no chances on the quality of the product. It will be noted that the Marinan Co., like the Rae Company, fined recently for the same offense, is one of those that sell direct to the farmer. After all, it should surprise no one that a company indifferent to who uses its product or how it is used should be equally indifferent to its quality, or contrariwise that firms that permit their serum to go into skilled hands only should also guard jealously the high quality of their products.

Hall Dies in Train Wreck

Arthur G. Hall, president of the New York State Veterinary Medical Society, and vice-president and general manager of Borden's Guernsey Farms, Inc., was killed in a train wreck April 19. Doctor Hall was born in 1882 and was graduated from New York State Veterinary College at Cornell University in 1908. After a few years of teaching and of private practice, he joined the Borden organization in 1911. Recognized as an authority on milk production, Doctor Hall served for 20 years as a director of the Certified Milk Producers' Association of America, and operated his own certified dairy farm at Earlville, N. Y., apart from his duties with Borden's. He served as associate chief veterinarian of the Borden exhibit at the New York World's Fair, and is shown here inoculating a calf born at the Fair. Doctor Hall was a member of Alpha Psi. He is survived by his wife, Mrs. Bertha C. Hall.



Distinguished Research Worker Dies

Sir Patrick Laidlaw died at his home in London March 19, 1940.

Sir Patrick, whose name is intimately associated with canine distemper prophylaxis, had a long record of achievement in biological research when in 1923 he, with Major G. W. Dunkin, was selected to undertake a study of canine distemper made possible by the "Field" Distemper Fund. The success of that investigation is known to every veterinarian in this country. In 1931 he was elected an honorary fellow of the New York Veterinary Medical Society in recognition of his contribution to the available fund of veterinary knowledge and to the control of canine distemper in this country.